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Berne, Switzerland, 09/05/2001

Review of EMF Risk Evaluation and Policy Options Document

Dear Colleagues:

The printed third draft of your EMF Risk Evaluation and Policy Options Document arrived here (by surface mail) on August 20, 2001, which gave me little time for detailed reading. However, it is with great interest and enthusiasm that I first browsed through this impressive piece of work and then read in detail parts of it. Time did not permit me to read it all, but I think that I am able to respond to your general questions and to comment on one of the chapters (leukemia).

Before responding to your general questions, let me comment that I highly appreciate the logical and disciplined way, in which you have analyzed the issues involved in evaluating the practical significance of a wealth of scientific work, which in the past had been undervalued. As your work shows, the reason for this is because earlier evaluations had been on the basis of an inappropriate set of criteria.

Questions about Risk Evaluation

Question 1.

I fully agree with your position that theories should not be used to discount evidence, in particular if it is well documented and consistently found. In the interplay of theory and practice, such as has been typical in physics, theory has to be consistently reformulated to take into account new evidence. Otherwise we do not deal with science, but with religious or esoteric belief systems.

Question 2.

The notion of initial degree of confidence is interesting and useful, because it aims at taking into account the tendency to accept harmfulness, which I guess is a personality characteristic. It is difficult to say what the personality characteristic really reflects, but it must have to do with mental flexibility and openness. The effect of 'a priori' expectations is comparable to that of theory (Question 1), because the more strongly a person leans toward a preconceived notion, the more resistant he or she may be to accept evidence as convincing.

But looking at the three reviewers' reasoning, personality characteristics seemed less influential than scientific argumentation. Considerations explicitly (reviewers 1 and 2) or implicitly (reviewer 3) related to the evolutionary

development seem particularly convincing to me. An important thought is that of reviewer 1 relating to the small number of persons likely to be affected. It relates to that of reviewer 2 noticing that electric and magnetic phenomena are involved in normal physiology and thus might on the one hand be tolerated, but on the other hand might interfere with it and lead to human pathology.

A problem with this analysis of prior degree of confidence is that it had to be judged at a moment, when the reviewers most probably had already had some notions about observations of health effects.

As far as I am concerned, it seems too difficult for me to rate myself quantitatively, and I will give a qualitative answer. I was very doubtful about any ill effect of EMF before I started working in this field, and I also conveyed this view to friends who told me about their own experience of exposure and its alleged effects. My own research in this field started because I was asked by a government agency to do so, and not really out of my own interest, and at that point I was still very doubtful about any biological EMF effect. Then, only when our data from several sub-studies (on RF-EMF and sleep quality) showed the same pattern, did I develop more openness toward this question. The literature on EMF and melatonin, and later on leukemia further influenced my position.

Question 3.

I agree. Mechanistic explanations seem to me to represent a vague form of theory (Question 1), whereas epidemiology provides evidence. Your domino-metaphor (page A-22/A-23) is very helpful in understanding this. In the past, I have used a black box metaphor, where epidemiology examines the association between inputs and outputs of a black box and leaves the study of the contents of the black box to subsequent research, which may be mainly on cell or animal models. The domino metaphor is more differentiated and therefore, more helpful.

By the way, it has often seemed to me that laboratory scientists have found it easy to generate mechanistic explanations for their findings, whatever their observations may have been. Only, the resulting theories become less and less parsimonious.

A last comment to this question (which may reveal that I myself tend to follow this pattern as well) is that I have been wondering, why your reviewers were not more positive about the possibility of a causal chain of EMF leading to changes of cell permeability (Ca^{++} efflux), reduction of melatonin excretion and loss of control over cell reproduction (i.e. malignancy). There seems to me to be some logic to this sequence, but I would agree that in view of present evidence, this may still be classified as speculative.

Question 4.

To the extent that I have seen the literature on animal pathology, I agree with you. The animal studies I found were usually anecdotal, and observations in control animals were in general not reported. Therefore there would not be good reason to give much weight to the available reports. If you add that reports of animal pathology rather increased the degree of confidence, I interpret this as being based on the fact that whenever anecdotal studies are published they usually provide some level of evidence in favor of an effect rather than against. Is this, what you meant? Wouldn't this more likely simply express publication bias?

Question 5.

I agree with your statement that not all epidemiologists would be impressed by relative risks between 1 and 2, and I also agree with what seems to be your own position that an increase of risk by say, 20 to 60 percent should be taken seriously, if the studies have been done properly. The best known example of a risk increase of 20 to 30% is for passive smoking and lung cancer, and it seems to me that studies on EMF and childhood leukemia show even more variability in design, which, as you state correctly, increases the credibility of this association being causal.

Question 6.

Yes, I agree again. If there is a mechanism involving a reduction of anticarcinogenic activity (such as could be the case with a reduction of antioxidant activity), several if not all cancer subtypes could be affected. This is not comparable to local exposure to specific carcinogens such as those leading to lung or stomach cancer, or to tumor promotion by female hormones leading to gynecological cancers.

Question 7.

Having read Chapter 8 on Epidemiology of the Leukemias in more detail than the other chapters, I respond with reference to this particular chapter. I think that you have done an outstanding job of reviewing each single criterion of causality (Chapter 8.2), and I have nothing to add. Your arguments against causality and for causality are well thought through, and your 'Comment and Summary' part is clear and equally convincing.

Given my general and enthusiastic consent, I will not continue by making more detailed comments on particular chapters and lines of the text. I hope that this will be acceptable to you.

Question 8.

The overlap at 98 and 2% do not bother me, but the one at 50% seems awkward. A possibility would be to create a new category at the center of the scale, perhaps for the confidence range of 45-55%. But the disadvantage would probably be that it would give undecided reviewers a chance to avoid a clear statement.

As someone from a non-English speaking country I was wondering whether your use of vocabulary is easy to understand for people with little education. How would the following alternatives be?

Current phrase	Suggested alternative
Virtually certain	Almost certain
Highly probable	Very likely
Possible >50%	Quite possible
Possible <51%	Possible
Very improbable	Very unlikely
Virtually certain that it is not causal	Almost certain that It is not causal

Please excuse me for not going into more details. Unfortunately, I received the documentation late, and tomorrow will be my last day before departure to a congress in China.

Nevertheless, I hope that these comments will be of use to you.

Please give my warmest regards to Ray Neutra.

Sincerely yours,

(not signed; sent by e-mail)

Theodor Abelin, MD, MPH
emer. Professor

September 9, 2001

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Re.- Draft 3: An Evaluation of Possible Risks from Electric & Magnetic Fields from Power Lines, Internal Wiring, Electrical Occupations & Appliances

Dear Ray:

Thank you for the opportunity to review this Draft. I have spent much time examining it in the past two months, and I regret that my continuing health problems have delayed a response until close to your deadline.

I respectfully submit some general comments and responses to your specific questions before proceeding to some listed comments.

General Comments:

1. Document Format: I have in mind that the primary intended use of this Report will be by legislative analysts, cognizant State and Federal Agencies, local public health authorities, public stakeholders and by technical and scientific media groups.

   After careful reading and much reflection, I have concluded that the Report will be of limited value to its most important intended recipients in its present format. Even for those like us, with many years of close acquaintance with the many byzantine facets of this complex and still evolving field, it remains extremely difficult to acquire from this Report a comprehensive overview, to "get the big picture", even after multiple readings.

   As a specific example, to conclude that a particular exposure may carry "a 50-90% risk, or no risk at all" may be semantically correct, but I respectfully submit that it may involve administrative and political suicide to allow motivated politicians and administrators to indulge their inevitable feeding frenzies over such statements, which occur in most sets of evaluations.

2. You are no doubt aware that this is a controversial decision. It should be justified by a detailed account of the history of Bayes' theorem, and its strengths and weaknesses when applied in the present Report. This should be a major opening argument. Yet I do no not find such a discussion anywhere in the Report. A recent evaluation of Bayesian methods succinctly stated:

   > "At the core of Bayes' long-delayed popularity is an admission that the world is rife with uncertainty and often not suited to clean statistical tests. When repeated experiments are not feasible, Bayesians believe

that sweeping generalizations should not rest on a small amount of new data. Instead they would bring to bear knowledge accumulated over many years, assign a weight to each part of that knowledge and then consider the new data."

A contrary position has been expressed by Richard Lowry, a Vassar psychologist and Bayesian skeptic:

"The intuitive nature of Bayesian methods adds little to their appeal. By assigning mathematical weights to subjective beliefs, Bayesians corrupt their data. The frequentist would conclude that no answer at all would be better than one lacking investigators' confidence."

As pointed out by M.L. Jordan at UC Berkeley:

"The result of a Bayesian calculation is usually not a single number but a series of probabilities. Part of the Bayesian spirit is that there is always uncertainty about everything, and this uncertainty should always be reported."

These statements would appear to epitomize the *modus operandi* adopted in the Report. To the academic purist, they may justify the disappointingly discursive and seemingly inconclusive findings. But to CEOs of power utilities, to State and Federal environmental planning agencies, and to public health authorities, and to all others lacking sophisticated statistical experience, they may become the ultimate frustration.

3. RESPONSES TO SPECIFIC QUESTIONS

Q.1 Do you agree that we should not be greatly influenced by arguments based on physics and simplified biological models suggesting that residential and occupational levels of EMFs cannot possibly produce bioeffects?

I agree. Objections from physicists arise in equilibrium thermodynamics involving a basic tenet that perturbations with energies below atomic thermal collision energies cannot be effective stimuli. There are many observations to the contrary in biological systems, as for example in the ear, where the auditory threshold involves a receptor displacement of 10^{-11} meters, or the diameter of a single hydrogen atom and substantially below the collision energies of receptor atoms and molecules. The doctrinaire attitude of these physicists is that, "Since your observations do not fit my models, therefore they are artifacts."

Similar objections from biologists also typically arise in inappropriate models of threshold sensitivities, based in equilibrium thermodynamics and ignoring the overwhelming evidence for nonequilibrium, nonlinear organization in biosystems. This involves coherent energy states and highly cooperative transitions, with good and growing theoretical and experimental evidence for sensitivities below thermal thresholds, and very importantly, with evidence that tissue sensitivities are set by populations of elements and not by a single receptor.

Q.2 Do you concur with the three core reviewers that their degrees of confidence In various relative risks are constrained by lack of dramatic changes in disease rates after the introduction of electricity and as the use of electricity has increased?

I disagree. There is at least one excellent study not cited in the Report relating residential electrification to the emergence of the childhood leukemia peak:

Milhain S, Osslander EM. Historical evidence that residential electrification caused the emergence of the childhood leukemia peak. *Medical Hypotheses* 56(3):290-295, 2001.

The authors conclude, *inter alia,* that "During 1928-1932, in states with above 75% of residences served by electricity, leukemia increased with age for single years 0-4, while states with electrification levels below 75% showed a decreasing trend with age (P = 0.009). During 1949-1951, all states showed a peak in leukemia mortality at ages 2-4. At ages 0- 1, leukemia mortality was not related to electrification levels. At ages 24, there was a 24% (95% confidence interval, 84 1 %) increase in leukemia mortality for a 10% increase in the homes served by electricity. The childhood leukemia peak of common acute lymphoblastic leukemia may be attributable to electrification."

These data should not be ignored. The position stated in your question appears to be a perpetuation of the ancient shibboleth perpetrated by the physicist Jackson at UC Berkeley in his 1991 paper published with the imprimatur of the National Academy of Sciences.

Q.3 Do you agree that lack of a mechanistic understanding should not decrease confidence in the epidemiology, since lack of a mechanistic understanding is not sensitive or specific?

I agree. To the extent possible, the epidemiology should stand as an independent body of evidence. Moreover, as in the case of cigarette smoking and lung cancer, public health policy should not await arrival of a complete body of scientific knowledge before establishment of essential health safety guidelines. However, there comes a point where further epidemiological studies are fruitless ***unless they take full account of new and growing mechanistic knowledge.*** We appear to be approaching that guideline in planning future research.

The importance and validity of mechanistic knowledge is exemplified in the following example of the progression of new knowledge on the interaction of 60 Hz magnetic fields with inhibition of breast cancer cell growth by melatonin. The initial observations by Liburdy of inhibition of the melatonin antiproliferative action by 1.2 μT 60 Hz fields in 1993, has been confirmed and extended by three laboratories (Blackman et al., 1998; Luben et al., 1998; and Morris et al., 1998). The most recent study by Ishido et al., (Carcinogenesis 22(7): 1043 -8, 2001) confirms the previous work and provides *evidence for uncoupling of signal transduction from melatonin receptors to adenylyl cyclase.*

Studies of this type reach to the very apotheosis of a mechanistic understanding. They give the lie to the epidemiologists' comfortable notions that it is appropriate to dismiss mechanistic studies as "insensitive and nonspecific."

Q.4. Do you agree with the view that the animal pathology literature is largely null, and that this pattern of evidence should not pull down our degree of confidence in the epidemiological literature?

I agree.

Q.5. Do you agree with that position that relative risks between 1 and 2 should be taken seriously unless there is specific evidence for confounding or bias?

I agree. In this regard, I suggest that the Report does not appropriately consider, at least from a theoretical viewpoint, the fact that in civilized societies an unexposed population no longer exists. As is the case for ionizing radiation, there may be no threshold with respect to cumulative dose; and as Hoteling at UC

Berkeley pointed out many years ago, low level exposures that are potentially pathogenic then become immersed in a sea of other low-level competing factors.

Q.6. Do you agree that a lack of specificity in the association of EMFs with subtypes of cancer should not reduce, and might even increase, the degree of confidence in causal associations between disease X and EMFs?

I agree. But the question of EMF relationships with a variety of subtypes of cancer again raises essential questions about ***mechanistic interventions.*** At issue is whether EMFs may be a significant co-factor in aspects of carcinogenesis. Occupational **exposures** to high magnetic field levels along with metallic oxide fumes has been cited in occurrence of non-Hodgkin's lymphoma and immunosuppression in aluminum smelter workers (Davis and Milham, 1992). Weedicides and pesticides may be similarly involved, and in our domestic environments there are a myriad potential carcinogens.

Q.7. Is the draft adequate in presenting arguments for and against causality, or are weak arguments assigned to the "con" and "pro" positions"?

I have given much thought to this question, because the ultimate evaluation of the historic value of the Report rides on the answer. I conclude that, in a clear attempt to avoid even a hint of reviewer bias, the preparers have failed to act as an independent committee of experts. I submit that this should be their clear and essential role. That is what is expected of them by involved organizations and individuals. Without it, I am mindful of Hamlet's tragic musings:

"The native hue of resolution is sicklied o'er with the pale cast of thought."

Thank you for the opportunity to review this draft. I have found it an interesting and challenging experience.

Sincerely,

W.Ross Adey, M.D.
Distinguished Professor of Physiology

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August 31, 2001

Dr. Raymond Neutra
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Dear Dr. Neutra:

I am writing to provide public comments on the California Department of Health Services (CDHS) *draft* Report entitled, "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances." The views expressed in this letter are solely my own and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas & Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District and San Diego Gas & Electric Company.

I would like to begin by giving some background about myself. I obtained a bachelor's degree in mathematics form Cairo University in 1959, a master's degree in statistics from the University of Chicago in 1962 and a Ph. D. in statistics from the University of California, Berkeley in 1965. I joined the faculty of the School of Public Health at UCLA in 1965 where I continue to be a full professor of Biostatistics. In addition, from November 1985 until June 2000, I served as the Dean of the School.

Over the years, I have carried a full load of teaching and research in biostatistics and public health. I have also developed specific expertise in the area of potential health impacts from extremely low frequency electric and magnetic fields (EMF). This has resulted partly from my serving as consultant to the Electric Power Research Institute (EPRI) from 1987 to 1997 and being a member of their advisory committee on EMF from 1987 to 1994. In that capacity, I assisted in the interpretation of the available scientific literature in an effort to assess the current state of knowledge about health impacts from long-term exposures to EMF. Part of my responsibilities was to help EPRI formulate EMF research priorities. In addition, I participated in the peer review process of applications for funding as well as in site visits to review progress of funded research. My own research in the field is summarized in the following two publications:

Kheifets, L.; Afifi, A.A.; Buffler, P.A.; Zhang, Z.W.: "Occupational Electric and Magnetic Field Exposure and Brain Cancer: A Meta-Analysis." Journal of Occupational and Environmental Medicine, 37:1327-1341, 1995,

Kheifets, Leeka I.; Afifi, A.A.; Buffler, Patricia A.; Zhong, Zhang, W.;
Matkin, C. Chantel: "Occupational Electric and Magnetic Field Exposure and Leukemia." Journal of Occupational and Environmental Medicine, 39:1074-1091, 1997.

In these papers, I served as the senior statistician and wrote parts of the manuscript. Since that time, I continued to have an active interest in the area, including following the literature and attending some relevant meetings.

In preparation for writing these comments, I reviewed the CDHS *draft* Report on EMF as well as several other scientific papers and summary reports, including:

> IARC Working Group, Day, N., Chairman, June 2001. IARC Staff Summary for Monograph 80: "Extremely Low Frequency Electric and Magnetic Fields." International Agency for Research on Cancer, Lyon, France.
>
> Advisory Group on Non-ionising Radiation, Doll, R., Chairman, 2001. "ELF Electromagnetic Fields and the Risk of Cancer, Report of an Advisory Group on Non-ionizing Radiation," Volume 12, No. 1, National Radiological Protection Board, Chilton, England.
>
> NIEHS EMF-RAPID Program Staff, 1999. "Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields." National Institute of Environmental Health Sciences, Research Triangle Park, United States.

There has been substantial public and scientific interest since Wertheimer and Leeper's 1979 paper suggested a possible association between residential exposure to extremely low frequency electromagnetic fields (ELF EMF) and leukemia or brain cancer in children and adults. Several private and public organizations have made research funds available in an effort to clarify whether such an association exists and, if it does, to quantify the effect. There is a large number of scientific reports available to assess whether EMF presents a potential public health risk. These reports describe results from laboratory experiments, whole animal bioassays, and epidemiological studies in both residential and occupational environments. The size and complexity of this literature are adequately described in the CDHS *draft* Report on EMF.

I would like to make three points. First, the CDHS *draft* Report on EMF overestimates the potential EMF risks. Second, a CDHS report that incorrectly assesses public health risks can have adverse consequences. Third, it is important that the CDHS correct the report so that the general public can better understand the current state of knowledge and so that the relevant government agencies can establish appropriate EMF policies. Elaboration of these points follows.

1. On page 1 (lines 7-10), Executive Summary, 'Statement For The General Public', the CDHS *draft* Report on EMF states:

> "It is 'more than 50% possible' that EMF at home or at work could cause a very small increased lifetime risk of childhood leukemia, adult brain cancer, and Amyotrophic lateral sclerosis (ALS, Lou Gehrig's Disease). As this phrase implies, there is a chance that EMFs have no effect at all."
>
> "It is 'more than 50% possible' that EMF at home or at work could cause a 5-10% added risk of miscarriage, and again, as this phrase implies, there is a chance that EMFs have no effect at all."

However, most scientists today would probably not agree with these assessments. For example, the NIEHS EMF-RAPID report states in its Executive Summary:

> "Virtually all of the laboratory evidence in animals and humans and most of the mechanistic work done in cells fail to support a causal relationship between exposure to ELF EMF at environmental levels and changes in biological function and disease status."

That report goes on to state:

> "The lack of consistent, positive findings in animal or mechanistic studies weakens the belief that this association is actually due to ELF EMF, but it cannot completely discount the epidemiological findings"

It is largely on the basis of the epidemiological findings that the CDHS *draft* Report on EMF bases much of its conclusions. I agree with the current draft that the epidemiological data on childhood leukemia raise potentially important scientific questions that need to be addressed in future EMF research efforts. There are, however, more appropriate ways to describe the remaining scientific uncertainty. For example, in the cover letter to the NIEHS EMF-RAPID report, the NIEHS Director concluded that:

> "The NIEHS concludes that ELF-EMF exposure cannot be recognized at this time as entirely safe because of weak scientific evidence that exposure may pose a leukemia hazard"

In Britain, the NRPB described the remaining scientific uncertainty by concluding:

> "After a wide-ranging and thorough review of scientific research, an independent Advisory Group to the Board of NRPB has concluded that the power frequency electromagnetic fields that exist in the vast majority of homes are not a cause of cancer in general. However, some epidemiological studies do indicate a possible small risk of childhood leukemia associated with exposure to unusually high levels of power frequency magnetic fields."

In summary, my opinion, based on the state of knowledge today, is that the CDHS *draft* Report on EMF presents an overestimate of the potential for EMF risks.

2. A CDHS report that incorrectly assesses public health risks can have adverse consequences.

It is important for the CDHS *draft* Report on EMF to correctly assess the available scientific information and perform a risk assessment that reflects the current uncertainty about any public health risks. While negative impacts on Californians can occur from either an under-estimation or an over-estimation of EMF risks, I believe that the current CDHS draft errs on the side of over-estimation. An overestimation of the effects of exposure to ELF EMF and efforts to warn the public about such possibly exaggerated effects can create unnecessary fears and may thus have a negative impact on the population, quite the opposite of what may be intended.

Sometimes, the case of EMF is likened to that of smoking: in both cases the actual mechanism linking the exposure to the condition is not identified. However, there is a very clear difference: in the case of smoking, the statistical associations observed in epidemiological studies are large and compelling, whereas, in the case of EMF, the observed associations are weak and debatable. In addition, the available information from the epidemiological studies on EMF is not supported by the literature on laboratory experiments and whole animal bioassays (where formal National Toxicology Program protocols were followed to assess cancer risks for rats and mice from lifetime exposure to high levels of sixty-hertz magnetic fields).

I believe that the substantial efforts that were devoted to research since 1979 have not shown that exposure to ELF EMF can produce negative health effects that are of concern at a public health level. Indeed, other public health problems provide a much more compelling case for public and private funding. For example, the conditions most implicated by EMF research, namely brain cancer and childhood leukemia, could benefit greatly from a concentrated effort of attempting to find truly potent risk factors and, if possible, ways to modify them. From a broader perspective, a number of other public health issues are crying out for funding. These include, but are not limited to, violence prevention, prevention of injuries, both domestic and occupational, care for the elderly, prevention of substance

abuse, as well the vast area of how our behavior, including what we eat and how we spend our leisure time, affects our health and how such behavior can be beneficially modified.

Furthermore, if any of the observed EMF associations are in fact causal, most scientists would agree that such effects are small. For example, in the case of childhood leukemia, it seems that there is no association with exposure to ELF EMF at a level lower than 0.2 microtesla. The observed statistical association is with exposure to levels higher than 0.4 microtesla, a level to which only a very small fraction of the population is exposed. Based on pooled data from a number of scientifically rigorous studies, it is estimated that the fraction of risk of childhood leukemia attributable to exposure to ELF EMF is 4% with a 95% confidence interval of –5% to 12% (see Greenland, Sheppard, Kaune et al., 2001, "A pooled analysis of magnetic fields, wire codes, and childhood leukemia," Epidemiology 11:624-634). This wide confidence interval is an indication of the high levels of uncertainty that characterize most research in this area. Current research indicates that our knowledge is even less certain with regard to the other conditions cited above.

The key point is that Californians look to the Health Department for credible information about public health risks. We each have limited resources and attention, and it is important that these are focused on issues that impact our individual and families' health and safety.

3. It is important that the CDHS correct the report so that the general public can better understand the current state of knowledge and so that the relevant government agencies can establish appropriate EMF policies.

The CDHS *draft* Report on EMF should be revised to support meaningful public communication and education. When modified, this report will form a better basis for evaluation of appropriate policies for funding additional EMF research and taking precautionary steps in the face of the remaining scientific uncertainty. The California Public Utilities Commission, other state agencies, as well as the general public need a report that is technically sound, that represents the current scientific understandings, that communicates the key issues in a fair and balanced manner, and that can be used to support the evaluation of appropriate statewide EMF policies.

I appreciate the opportunity to provide these comments.

Sincerely,

Abdelmonem A. Afifi, Ph.D.

October 22, 2001

Dear Raymond!

I have now at last looked at your risk evaluation material and I am duely impressed. Unfortunately time does not allow me to read it in the detail that it deserves so I will only give some general comments.

There is no question in my mind that this is by far the most comprehensive and amibtious risk evaluation on this topic that has been conducted. Not only is it comprehensive but also intelligently done. I also find that the process has been very carefully thought out with possibilities for all parties to participate and with a great openess. I realize that there has been comments on various aspects of the process, such as the choice of three assessors, but I am convinced both that you choose your methods very carefully and that whatever alternative you would have chosen had also generated comments.

The Bayesian approach that you have chosen is one basis for your risk evaluation. It is not possible for me to evaluate this procedure and its various inputs. Without being an expert, I would think, however, that this method is a rather robust one and not too sensitive to alternative inputs or techniques.

In your letter of July 9 you ask certain specific questions. I fully agree with 1; can not answer 2; agree fully with 3; can not answer 4; fully agree with 5; partly agree with 6; yes on 7; terminology is very difficulyt as you state in 8 and your approach is very good, still I think that these terms are virtually impossible to give precise meanings.

Bayesian Net to combine all the estimates of bias confounding chance and causality needs to be done. He is being paid $40 k by EPRI to do this but it wont be done until after we are finished. We had said in our guidelines that we would not do this because it would be too opaque for others to evaluate.

Best regards and congratulations to a successful work!

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RE: Review of "Electric and Magnetic Field (EMF) Risk Evaluation and Policy Options"

Dear Sir:

I am pleased to provide a review of the third draft of the above report. While my perspective is that of a public health physician epidemiologist and thus come from a "prior" quite similar to your three primary reviewers, I also asked John Lorenz, one of our senior nuclear engineers familiar with EMF issues to review it as well. The comments below combine our two perspectives.

I would like to first congratulate you for preparing an excellent evaluation document. Your thorough review and analysis adds perspective and insights to the controversy surrounding EMF. It is among the best analytic presentations of the literature I have seen and aside from its utility to California as the basis for policy options, it will be of considerable utility to health departments such as Wisconsin's. I have not had the opportunity to use or assess the California Risk Evaluation Guidelines so they were a bit foreign to us and its "degree of confidence" approach was at times confusing. We comment on that later. The arguments and positions taken were well presented, clearly articulated and the decision processes transparent.

There are several features of the Evaluation that are particularly valuable.

1. An important point you stress repeatedly through the Evaluation is that real world EMF exposure involves a complex mix of frequencies, modulations, intensities and temporal variations as well as possible contact exposures. The complexity of the alleged agent is important to consider in analysis of research findings and in policy discussions.

2. The discussion of the four policy frameworks from which the Evaluation can be viewed is valuable. It is important to remember that our different views on policy may result from beliefs more fundamental than knowledge of the available data.

3. The recommendations for additional research contained in the conclusions section (p.318) are specific and are targeted toward clarifying questions that have confused the relationship between EMF exposure and the identified diseases.

We also feel that several areas in the Evaluation need clarification or change.

1. There is a bias in the report toward heavily weighting epidemiology study findings. This is not surprising since the reviewers are public health epidemiologists or, if not epidemiologists, part of the California Department of Health. The epidemiological evidence is used as the gold standard throughout the Evaluation and evidence is likely to be disregarded if it conflicts with or does not support the epidemiology associations. As a case in point, epidemiological evidence is cited as the basis for the assumption (p.57, Table 6.2.2. (F2)) that there is no additional risk above 8-10 mG. This assumption is used as a basis for explaining the lack of positive animal studies that use small numbers of animals at high intensity fields. Such an argument helps unify the disparate findings, but is still largely hypothetical. What is the basis for the "no additional

risk" assumption when the mechanism of effect is unknown and, individually, most epidemiological studies are just barely or not statistically significant and have insufficient numbers at the higher doses to expect to demonstrate a dose-response? (Relates to your questions 1, 3, 4 and 7)

2. Determining the prior degree of confidence, while an interesting exercise did not seem to be used or particularly helpful in this evaluation. The advance to greater confidence in causality from the "prior" does not appear to be used. If you fully followed the Bayesian method of analysis, there surely would be value in determining the prior of each of the reviewers. However, in this evaluation, it seemed that the prior was gradually discarded as other streams of evidence were considered, rather than modified by the subsequent evidence. In addition we question the ability of even a trained scientist to ignore a lifetime of experience when determining the degree of confidence he or she would have had without the lifetime of experience. (Relates to your question 2) Perhaps a better measure would have been determining the position of each of the reviewers vis a vis the strength of their causality confidence based on their reading and study prior to the comprehensive, systematic review of all the literature that was done. This assumes they had not already read and assessed all the literature included in the review. In other words were their current (preview reviewer status) beliefs concerning EMF disease causality changed by the months long comprehensive review?

3. The exact meaning of "degree of confidence" should be better defined. Section 1 extensively discusses what the concept is not. The discussion is valuable, but there should also be a clear definition of what degree of confidence really is, how it is derived and its significance. Is it really an attempt to objectify what used to be called "professional judgement"? Perhaps because we are not familiar with applying the California Risk Evaluation Guidelines, our group members reading the report did not all come to the same understanding of the term's meaning from reading the Evaluation. Is it a degree of confidence that the agent is a causative factor for a disease, or is it a degree of confidence that the agent is causative at a specific level (e.g. RR = 1.2)? Spontaneous abortion is a good example where one might be very confident that the epidemiology supports causality, but not confident that the extent of the impact described in the epidemiology studies is accurate. Your discussion seems to imply that a 20% degree of confidence means a reviewer feels the non-causation probability is five times the causation probability. Is this a correct interpretation? The fact that the term is similar to the familiar statistical "confidence limits" only added to our confusion. In fact it appears that each reviewer put "confidence limits" on their degree of confidence. Our discomfort with this terminology and its strong subjective component is in part due to our lack of experience using such an approach. An additional explanatory section in Section 1 would be useful. Is this an attempt to come up with a semi-quantitative approach to professional judgement? Perhaps changing the term itself would help – but we can't think of anything better.

4. We have difficulty supporting using the degree of confidence as a multiplier in the economic analysis to determine how much morbidity and mortality can be avoided by removing a risk factor. It would seem better to simply use the statistical confidence interval from either the meta-analysis or the key study used to estimate the population impact. On page 315, lines 41-55, the discussion includes using one's degree of confidence in causality in order to derive expected mortality numbers. The example uses a 20% degree of confidence. This is a meaningless exercise, since the reviewer's confidence is only related to whether the agent is causative and not to the amount of disease it causes. When the reviewer feels that the probability of non-causation is five times higher than the probability of causation, wouldn't it be more appropriate to assume removing the agent will not reduce mortality numbers? If the disease is very prevalent, take actions that will remove uncertainty from the analysis rather than using risk numbers that have been modified by the degree of confidence. At the very least, this use of the degree of confidence multiplier might best be restricted to degrees of confidence over 50%.

5. The term "Possible >50%" could be replaced by "Probable" and the term "Possible <51%" could be replaced by "Improbable but possible".

The Evaluation is an excellent review by three experts. They have drawn conclusions based on their review and their background knowledge. They have made their determinations very transparent and understandable. They seemed reasonable and well considered. We felt it was appropriate to comment on the method they used in reaching their conclusions, or any factual errors that may have entered into their considerations. However, we don't feel it is appropriate to comment on their individual conclusions. We did not have time to review all the information as thoroughly as they did and thus whether we agreed with them or not would be more due to our "priors" rather than our systematic analyses using your methodology. Barring methodological changes or factual corrections, their conclusions should not be changed for the final version of the Evaluation.

If you have questions about our comments, please contact me at (608) 266-1253 or anderha@dhfs.state.wi.us

Congratulations on a job well done. Please send me a final copy when you complete the "draft 3" review.

Sincerely,

Henry Anderson, M.D., Chief Medical Officer
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RE.- ELECTRIC AND MAGNETIC FIELD (EW) RISK EVALUATION AND POLICY OPTIONS

Dear Mr. Collins,

Dr. Raymond Neutra sent me this document to review. I will start with my responses to your eight leading questions:

1. I agree that arguments based on physics and simplified models should be disregarded. I was Vice-Chairman of a Science Advisory Board Committee to critique an EPA report on EMF; we heard evidence that there was clear evidence that weak 50 Hz fields could influence intact systems although several senior physicists told us that no tissue effects were possible. We were influenced from experiments on the effects of radar on bird migration-, on demonstrated effects in expediting bone healing; and on effects on fish. All of these demonstrated that the arguments from purer physics were not applicable.

2. Long term changes in disease rates are hard to interpret, but there is evidence that childhood leukemia might be increasing slowly. The point should be made that this disease is almost certainly niuitifactorial in origin and requires a combination of circumstances for its initiation. The question is whether EMF exposure increases the risk.

3. I agree with this position. Lack of mechanistic understanding is not a reason to question the epidemiological results if these show consistency.

4. I agree with this position.

5. I agree that relative risks between 1 and 2 should be taken seriously. The demonstrated effect of particulate air pollution on respiratory and cardiac mortality provide a recent example where the force of the evidence is now being acknowledged.

6. I agree with this position; the risk in relation to diseases other than childhood leukemia is difficult to define, and the emphasis in this document is, I think, correct.

7. Yes; the document does an excellent job of presenting the uncertainties fairly. In fact, I think it is the best risk assessment analysis of a low risk outcome that I have encountered.

8. The only correction I would suggest to this Table is to use the phrase "probable" in describing the risks with a confidence range of 50-90%

The only general comment I would make is that the multifactorial genesis of the outcomes under examination should be stressed more; and there should be a section describing the difficulties of assessing risk in conditions which are known to be affected by several factors.

I hope these comments are useful.

Yours sincerely,

Dr. David V. Bates, MD, FRCP, FF, FACP, FRSC
Professor Emeritus of Medicine

12 September 2001

Comments on the Third Draft of the Electric and Magnetic Field (EMF) Risk Evaluation and the Policy Options document written by DHS, DEODC, State of California.

by Carl Blackman*, US EPA, ECD (MD-68), Research Triangle Park, NC 27711-2055
* opinions are strictly my own and are not necessarily those of my employer.

Answers about Risk Evaluation:

1. I agree that physical theories regarding the likelihood that EMFs at residential and occupational levels could cause bio effects should not be used to discount evidence. To do otherwise would be contrary to use of the scientific method.

2. The reasons laid out by each of the three core reviewers to establish their initial (prior) degree of confidence that residential or occupational EMFs could produce relative risks of various sizes are cogent and convincing. My degree of confidence would increase as I gained laboratory experience testing the environmental/occupation agent. In the case of EMF, my degree of confidence is higher than any of the core reviewers because I have performed experiments with these specific EMFs since 1970. My research has found bio effects at very low intensities, e.g., my published work has shown effects caused by exposures as low as 0.2 mG and 10 V/m.

3. The core reviewers did not reduce their degree of confidence in epidemiology on the grounds that lack of mechanistic understanding is not sensitive or specific. I agree with this determination because EMFs are a complex assortment (cocktail) of possible causative agents and their individual influence on various components of cell biology leading to possible tumor formation is unknown.

4. The animal pathology literature is largely null effect, but the core reviewers did not let it reduce their degree of confidence in the epidemiological literature. I agree the animal literature should not have a negative impact on the epidemiology. In fact, it is possible to consider the animal literature, composed almost exclusively of exposure to sinusoidal fields of constant intensity, as supportive of more complex exposure paradigms which are closer to the actual agent in real life exposures captured by the epidemiology studies, whether it was measured or not.

5. Should relative risks between 1 and 2 be taken seriously? I believe they should because one does not know if the proper exposure metric is being tested in the particular study. It could be that the actual risk is generally low, or that a small fraction of the 'cases' are the only ones actually exposed to the active EMF components. Additional, focused research both in the laboratory and in the field is needed to establish probable metrics to evaluate.

6. Should the lack of cancer subtype associated with EMF exposure and evidence for effects on various types of disease reduce or increase the degree of confidence that epidemiological associations between disease X and EMF are causal in nature? I believe we don't know enough about how a series of EMF-induced biological perturbations, and the possible biological states of sensitivity, can affect tumor formation. Without such understanding, we can only monitor changes that we think detrimental. Evidence that EMF exposure is associated (see comments on Scarfi et al. results below) with specific diseases indicates multiple consequences can occur from EMF-induced changes. The reviewers are correct to have this evidence increase their degree of confidence.

7. Has an adequate job been done presenting arguments for and against causality? I think the argument can be made stronger for causality if the NIEHS document had not taken as the exclusive and thorough review of the literature up to 1998. Other exhaustive reviews, for example by US Environmental Protection Agency and by NCRP, demonstrate consistent in vitro biological effects across laboratories together with demonstrations of unusual dose and frequency responses. Inclusions of these results would have strengthened the decision to prevent the lack of

animal results, obtained with constant intensities and frequencies, from reducing the degree of confidence in the epidemiology data, obtained with complex exposures in the "real" world.

8. The plain language risk evaluation guidelines are not user friendly; how can they be improved? I think the present blurring of barriers between different categories is called for because the data do not allow for clear distinctions. Readers need to know that and learn to deal with it.

Specific comments on the Evaluation text.

page	table	line	comment
6		4-6	The NIEHS document should not have been taken as the exclusive and thorough review of the literature up to 1998. Other exhaustive reviews, for example by US Environmental Protection Agency and by NCRP, would have demonstrated consistent in vitro biological effects across laboratories together with demonstrations of unusual dose and frequency responses. Inclusions of these results would have strengthened the decision to prevent the lack of animal results, obtained with constant intensities and frequencies, from reducing the degree of confidence in the epidemiology data, obtained with complex exposures in the "real" world.
6->7		66->2	The MCF-7 cell responses have been replicated in two independent publications, Blackman et al. Bioelectromagnetics (22:122-128, 2001), and Ishido et al. Carcinogenesis (22:1043-1048, 2001). The paper by Ishido et al. also presented evidence that magnetic fields may cause uncoupling of signal transduction from melatonin receptors to adenylyl cyclase. Since adenylate cyclase is intimately involved in one of the several fundamental signal transduction processes, these results may have broader application as a mechanism for other EMF-induced biological effects. This information should definitely be added to the text.
7		67-71	It is unfortunate that low-frequency modulated radiofrequency research could not have been incorporated in the literature review because research published from the late 1970s through the 1980s on ELF and ELF-modulated RF have relevance to issues raised in the California evaluation. Essentially, they would have greatly enhanced the degree of confidence to ignore both the biophysics/mechanisms and animal data, and independently, to enhance the degree of confidence in the epidemiology data.
16		47-54	These lines comment on EMF-induced changes in blood serum levels of melatonin or of its metabolites in urine. There is no discussion about possible EMF effects on the action of melatonin in cells throughout the body. The MCF-7 papers indicate that this issue may be one of the missing links to form the connection between melatonin and EMF-induced abnormalities.
17		46	The 't' seems to be misplaced, and probably needs to be deleted. Otherwise, the copyediting in the text is outstanding.
18		29-33	As a general statement, the text is correct. However, it is all the more important to highlight the results of those studies that did look at different EMF exposure conditions. There are a number of studies that have tried to characterize the EMF field parameters that could lead to refined descriptions of dose response relations and thus stronger

			biological effect data. Our own lab focused on this area in the 1970s, 1980s and into the 1990s. One paramount observation that has been shown by many investigators (see work and reviews from the independent labs of Adey, Blackman and Liboff) is the fact that there can be many intensity regions, called windows, within which effects can occur and outside of which no EMF effects are observed. Similar, but less thorough experiments have demonstrated windows in the frequency domain and comparing sinusoidal with pulsed fields. Although no widely accepted underlying mechanisms have been demonstrated to be responsible for these phenomena, the observations clearly show that EMF may not cause changes in a dose-response manner that is similar to the responses observed for chemicals. Thus, had these reports been cited, they would have increased the confidence of the core reviewers that observations are paramount in evaluating EMF-induced phenomena.
19		30-32	As mentioned in my comments about text on page 18, there are studies showing effects in the 2-mG range and lower that could serve as one focal point to begin additional research.
22		42-52	This is a very interesting section, describing an issue that is at the core of a lot of the debate about EMF effects.
26		3-6	The situation may be more complicated that described here because the dose response may not follow a linear or monotonically increasing function. A case in point is the intensity windows reported by a number of investigators.
28		2-3	Good, concise statement.
28		31-34	The text gives a good citation and an excellent analogy.
28		37-38	Excellent statement. There are many examples in the biological literature to support this statement.
28		40-45	Excellent summary.
28		58-61	This fact, comparing anthropogenic EMF with background fields, is often ignored, especially in epidemiology when one is comparing differential results between two slightly different levels of exposure to EMF.
30		12-16	This is an excellent identification and enumeration of these facts that are associated with EMF exposure.
31		27-31	Comparing the loudness of a radio with the information content in the sound is an excellent analogy that is very applicable to EMF exposures.
32	5		The meaning of the symbols for High Wire code and High Measured Field is unclear. The work of Kaune is not available for this reviewer to evaluate. However, the Bowman et al. 1995 paper, based in part on the publication by Blackman and Most (Bioelectromagnetics 14:413-431, 1993), states the "... findings suggest that the risk of childhood leukemia may be related to the combined effects of the static and ELF magnetic fields." It is unclear to this reviewer why the text in this review associated with the resonance concept, in all its manifestations, essentially dismisses it as a possible causative influence in epidemiology results. Does the non-peer reviewed work of only one author cause the concept,

		developed by several authors based on substantive evidence, to be declared unimportant and not worthy of additional examination? I believe it is a mistake to declare it so, as is done in this document.
33	32-34	Nevertheless, the results of both studies directly demonstrate that TWA may be an irrelevant measure for some EMF-associated biological effects. I think the text tries to back away too much from the implications of the Lee and the Li results.
36	C5,A7,F7	There are results from three independent laboratories showing EMF effects at 12 mG on melatonin-inhibited MCF-7 breast-cancer cell-growth. This fact documents that theory has so far failed to be a useful predictor of effects at low intensities. Theory also fail to predict effects reported in from the mid 1970s through early 1990s by several independent laboratories, which replicated or extended each other, using very low field strengths.
36	F7	See comments above for C5, A7 & F7. Also, one study has shown exposure of chicken eggs during 21 days of incubation to 10 V/m (intensity in air) electric fields (intensity frequently found in residences) alone can cause changes in brain tissue associated with calcium ion flux [Blackman et al., Bioelectromagnetics 9:129-140, 1988.]. Thus, both low-intensity magnetic and electric components can cause biological changes.
36	C8	Excellent point; an unusual, almost prohibitive standard is being used to evaluate EMF-induced effects. This approach should not be allowed in matters of public health analysis.
37	T	Scarfi et al. (Bioelectrochem. & Bioenergetics 43:221-226, 1997) shows increased micronuclei formation in lymphocytes from patients with Turner's syndrome (only one X chromosome) when the cells are exposed to pulsed but not to sinusoidal magnetic fields. No effect of these treatments is seen in lymphocytes from normal patients. The response of lymphocytes from Turner syndrome patients demonstrates the existence of at least one genetic subpopulation with greater sensitivity to specific types of EMF exposure. There may be other sensitive subpopulations.
38	F2,C2 & 7-11	The MCF-7 cell responses of Liburdy et al. have been replicated in two independent publications, Blackman et al. Bioelectromagnetics (22:122-128, 2001), and Ishido et al. Carcinogenesis (22:1043-1048, 2001). The paper by Ishido et al. also presented evidence that magnetic fields may cause uncoupling of signal transduction from melatonin receptors to adenylyl cyclase. Since adenylate cyclase is intimately involved in one of the several fundamental signal transduction processes, these results may have broader application as a mechanism for other EMF-induced biological effects.
39	T	The work of Lai and Singh show that single and double strand DNA brakes in rat brains are enhanced after exposure to ELF and to RF fields. Additional DNA strand breaks, especially double strand breaks, can have detrimental consequences to the organism. Free radical scavengers prevent this increase in breaks. This work has direct implications for cancer induction, and should be referenced.
43	T 615	Boorman & McCormick et al. study: Subsequent evaluation of background levels of thyroid C-cell adenomas & carcinomas in the controls of 10 studies using the new NIH 2000 animal food diet, demonstrated that the increase observed in this study was significantly above expected values for control animals (information provided by C. Portier, at IARC meeting June 2001). This observation should no longer be treated as an anomaly.

56	C1		Joint paper by Loscher and Anderson (Anderson et al., Envirn Health Perspect 108:797-802, 2000) highlight the differences in materials and methods used in the two studies and the possible reasons for the different results. Included as differences are different substrains of Sprague-Dawley rats, sources of diet and DMBA, environmental conditions and exposure metrics. My conclusion is that the experiments by the two groups were not sufficiently similar to declare one was a replicate of the other; they were similar experiments.
61	T 6.2.12	C1,C2 & F1	Excellent observations.
305		22-25	See comments on MCF-7 cell growth in p 38, F2,C2 & 7-11.
306		2	It does not appear that resonance effects can be so quickly dismissed as a causative factor; this conclusion is attributed to Kaune and Bowman. The work of Kaune is not available for this reviewer to evaluate. The Bowman et al. 1995 paper, based in part on the publication by Blackman and Most (Bioelectromagnetics 14:413-431, 1993), states the "… findings suggest that the risk of childhood leukemia may be related to the combined effects of the static and ELF magnetic fields." It is unclear to this reviewer why the text in this review associated with the resonance concept, in all its manifestations, essentially dismisses it as a possible causative influence in epidemiology results. Does the non-peer reviewed work of only one author cause the concept, developed by several authors based on substantive evidence, to be declared unimportant and not worthy of additional examination? I believe it is a mistake to declare it so, as is done in this document.
306		14	It has been reported that electric fields at gradients found in homes, can cause biological effects. See p 36, F7, above.
317->8		15->8	Excellent overview of possible research topics.
318		43-46	Other cell culture systems in addition to the ones stated should be studied, particularly with respect to signal transduction changes, and cell growth changes associated with tumor formation and development. This should be indicated in the text.
318		51-52	Not only melatonin production but also melatonin ACTIONS in target tissues should be studied, as dramatically demonstrated by the MCF-7 cell experiments (see comments for p 38, above).

Comments on the Policy Options text:

page	table	line	comment
1		1-52	Good abstract of document.
3-4		42-33	Very good description of changes that could be made to the power grid.
6		6	Net currents is a problem in homes, in addition to schools.
8		42-62	Good descriptions.

General comment: this should be a very useful document for the PUC.

September 8, 2001

Review of EMF Risk Evaluation and Policy Options documents from the California EMF Program

by Joseph D. Bowman, PhD
Non-ionizing Radiation Section
National Institute for Occupational Safety and Health

with contributions from
Greg Lotz, Russ Savage and Heinz Ahlers

Introduction

The California EMF Project asked me to review their documents on EMF risk evaluation and policy options. I agreed to do so because I had attended an advisory meeting on their risk evaluation guidelines, and had been a member of NIEHS's 1998 working group on EMF health effects.

Whether EMF is a health risk has been a difficult public health issue because exposures are widespread but the scientific evidence is inconclusive. In 1996, NIOSH issued a fact sheet *EMFs in the Workplace* (1996) that said EMFs are not a "proven health hazard", but outlined methods for reducing exposure for those who wished to take precautionary actions. Recently, reviews by NIEHS, IARC, and the National Radiologic Protection Board (NRPB) in Great Britain have concluded that ELF-EMF is a possible carcinogen based on epidemiologic associations with childhood leukemia, but they found the evidence too weak to recommend exposure limits or propose regulations that would mitigate exposures. When evidence for other diseases was reviewed, it was found inconclusive. NIOSH reviewed the NIEHS reports (Portier and Wolfe, 1998; NIEHS, 1999), and had minimal criticisms.

The California EMF Program cover the same issues in their documents and uses their detailed review of individual papers. However, the California approach is more comprehensive and uses innovative methods to reach their conclusions. Although the California evaluation uses many standard techniques (*e.g.* the Hill criteria, the IARC process for assessing carcinogenicity, quantitative risk assessment, cost-benefit analysis), the overall framework uses an original form of Bayesian logic that synthesizes all kinds of evidence and expresses its conclusions as a probability that EMF is a risk factor.

With the help of other NIOSH scientists, I have reviewed the first nine chapters of the evaluation document, and the policy options document, and our specific comments are in the attached table. Answers to the eight questions asked by the California EMF Project are given below. In the Conclusions, we discuss the issues raised by the California reports, which are most important to occupational health.

Answers to the Questions from the California EMF Project

1. Q. We have taken the position that we are not greatly influenced by arguments based on physics and simplified biological models that suggested that residential and occupational levels of EMFs can't possibly produce bio effects. We say that theories should be used to predict results that are falsifiable and should not be used to discount evidence. Thus, our prior degree of confidence is not vanishing small. Do you agree? Please comment.

   R. Questions 1, 3 and 4 all deal with the difficult aspect of balancing mechanistic evidence from theoretical and laboratory research results (generally lacking evidence of EMF bioeffects) with the epidemiological research

results that provide evidence of EMF health effects. The low weight given to the absence of support from simplified biophysical models is reasonable.

2. Q. Each of the three core reviewers have laid out their initial (prior) degree of confidence that residential or occupational EMFs could produce relative risks of various sizes. These estimates are constrained by what we know about animal bioassays for cancer and by the lack of dramatic change in disease rates after the introduction of electricity and as the use of electricity increased. Reasons are given for these judgements. Do they seem reasonable? How much higher or lower would you're a priori degree of confidence be for any environmental/occupational agent? For EMFs? Why?

   R. The justifications for the prior distributions are reasonable. However, the other prior distributions that the authors discard do exist among EMF researchers. If the goal of Bayesian inference is to capture the consensus of the scientific community, the implications of these other priors should also be reported.

3. Q. We were not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects nor were we convinced of a chain of events that led to pathology. Yet we did not let this pull our degree of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment.

   R. We agree that the theoretical and *in vitro* mechanistic studies do not explain how EMFs in the environment could cause the reported health effects. In most cases, the *in vitro* studies were conducted in model systems that are not remotely related to cancer studies, and are done with EMF exposures far higher than found in homes and most workplaces. Even if the *in vitro* studies showing EMF bioeffects are accepted as valid, they would not necessarily support a chain of events that might lead to pathology. We agree that the epidemiologic data should have more weight in the overall interpretation of the literature than these *in vitro* data that are unrelated to cancer development. This position is consistent with the other EMF reviews like the IARC framework where "limited evidence" from epidemiology and "inadequate evidence" from animals is sufficient to declare an agent a "possible carcinogen". With EMF, the debate is mainly over the strength of the epidemiologic evidence.

4. Q. We viewed the animal pathology literature as largely null, with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment.

   R. We agree that the animal studies literature is largely null, and there is little there to suggest that EMF is a carcinogen. We do not agree that the breast cancer promotion studies or the chick embryo studies increase the degree of confidence in the epidemiological literature. All the studies reported by the Loescher group have limited internal consistency and therefore are not clear in identifying an EMF effect. This is particularly true in light of the Battelle failure to find similar results after an extensive effort to repeat these studies. Likewise, the overall literature on chick embryo studies is difficult to interpret because of the inconsistencies in the data across various studies. We do agree, however, with the interpretation that these data and the animal studies literature in general do not negate the findings of the epidemiologic data.

5. Q. Not all epidemiologists would agree with our position that relative risks between 1 and 2 should be taken seriously unless there is specified evidence for confounding or bias to explain it away. Do you agree? Please comment.

   R. I do not have enough experience with epidemiology to set a lower bound for reliable odds ratios. However, the relative risks of 1.2 from the meta-analyses on adult leukemia and brain cancer are not a scientifically

reliable basis for a finding of possible carcinogenesis, especially with this wide-ranging collection of study designs and exposure assessments (see more comments on these meta-analyses below).

6. Q. We said that a lack of specificity in the association of EMFs with subtypes of cancer and evidence for effects on various types of disease did not pull down our degree of confidence and might even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature. Do you agree? Please comment.

   R. The fact that the *in vitro* studies showing EMF effects involve primarily cell signaling and other epigenetic processes supports the interpretation that EMF might have effects that would be general on many disease processes and not specifically affect a target organ or disease. If these *in vitro* findings are confirmed and more robust evidence is developed to support them, the acceptance of associations with many different diseases is a reasonable position.

7. Q. Have we done an adequate job in presenting the arguments for and against causality or are we assigning weak arguments to the "con" or the "pro" position?

   R. This question is extremely broad and begs an extensive, point-by-point reply, which is not possible at this time. Although I criticized a few of your arguments in my specific comments, the document in general uses reasonable arguments to make both pro and con arguments as persuasively as possible.

8. Q. Our Risk Evaluation Guidelines (REGs) define some "plain language phrases" to express our degrees of confidence. However, when we actually applied them we found they were not problem free: (a) some of these phrases are not mutually exclusive. For example, "Possible >50%" overlaps "highly probable" and "virtually certain." "Possible <51%" overlaps "Possible >50%". In this case, the overlap is slight, but important, since it is about the "balance of probability." (b) These phrases are grammatically awkward and they are not really "user friendly." How could we rephrase them, without violating the spirit of the REGs? Please write any suggestions next to each phrase.

   R. The phrases "50-90% Possible" and "10-50% Possible" definitely need to be modified. They are awkward, and use "possible" to mean "probability". The global warming report by the International Panel on Climate Change (Reilly et al., 2001) did far better:

   >99% Virtually certain
   90-99% Very likely
   66-90% Likely
   33-66% Medium likely
   10-33% Unlikely, etc.

   For public health purposes, however, I would replace the IPPC's adjectives with the familiar IARC terms "Probable" and "Possible".

   If better risk categories are devised, I think the probability ranges can be useful in communicating with the public. People are used to hearing posterior probabilities in weather forecasts, so probabilistic risk evaluations would also be understood if similar language were used:

   > It is *possible* that EMFs at home or at work could cause a very small increase in the lifetime risk of childhood leukemia, adult leukemia, etc. Putting this scientific judgement into numbers, the chances that EMF exposures actually caused these diseases are between XX% and YY%. This also means EMFs may have no effect at all.

Conclusions

The greatest strength of the California risk evaluation and policy options documents are their thoroughness – covering all diseases linked to EMF and considering all the arguments raised for and against causality and government intervention. The policy options document completes the effort by carrying the risk assessment through to a probabilistic cost-benefit analysis. This report uses all the techniques of modern epidemiology, risk assessment, and risk management to provide a complete discussion of public health policy on EMF in the environment.

The greatest weakness of California's risk evaluation is using only three reviewers, all Department of Health Services employees who are epidemiologists familiar with the EMF issue. This is justified as "the usual practice when the Department is asked by another agency for a technical determination" (p. 5). Given the subjective nature of Bayesian inference, a larger committee of reviewers from more disciplines would give their conclusions more credibility. Since the evidence for a health risk comes from epidemiology, a committee with toxicologists and other disciplines would most likely arrive at a lower probability for the risks.

The subjectivity issue looms largest where the California report concludes that EMF is a credible risk where other recent reports find the evidence inadequate. The California evaluation is the first to find that EMF is a possible risk factor for ALS and adult brain cancer (>50% probability). Also, the California report joins the NIEHS working group in finding EMF a possible cause of adult leukemia, where the IARC and NRPB reviews do not. The California report explains these discrepancies as the result of the "quality of evidence" method used by IARC and the "degree of confidence" process in California (p. 312). I do not find the technical distinction to be a persuasive reason for accepting the California results. Unless the California report gives a stronger justification for the merits of its approach, these new findings of EMF risks may be dismissed as the opinions of three individuals.

Another major shortcoming is the report's heavy reliance on two meta-analyses of the occupational EMF epidemiology (Kheifets et al., 1995 and 1997) for its assessment of adult leukemia and brain cancer (Chapters 8 and 9). Although well done, these meta-analyses pool results from a wide variety of study designs (cohort, case-control, PMR) and exposure assessment methods (measurements and *a priori* assignments based on job titles). A diversity of study designs violates the mathematical assumptions behind meta-analysis, and makes its pooled risks unreliable. Their exposure-response analysis is particularly unreliable because the high exposure groups in some studies equal the medium exposure groups in others.

Far more reliable are pooled risks and exposure-response estimates for occupational EMF and cancer from the comparative analysis of four electrical utility studies (Kheifets et al., 1999). This study uses the primary data from four cohort studies with full-shift EMF monitoring, and finds stronger associations with magnetic field exposures than the 1995 and 1997 meta-analyses:

	Pooled RR for highest exposure (> 16 μT-yr)	Pooled exposure-response (OR per 10 μT-yr)
Leukemia	1.48 (0.96-2.30)	1.09 (0.98-1.21)
Brain cancer	1.85 (1.17-2.98)	1.12 (0.98-1.28)

The data from these four studies are all homogeneous ($p > 0.5$), and the pooled risks above have $p = 0.05$ in one-tailed significance tests. Consistent data, a significant exposure-response, and relative risks above 1.2 all strengthen the epidemiologic evidence for a causal association. Thus, the Kheifets comparative analysis provides the same kind of evidence with occupational EMF as the pooled analysis by Greenland et al. (2000) does for a causal association with childhood leukemia.

I suggest that the chapters on adult leukemia and brain cancer rely more on the Khiefets comparative analysis than on her meta-analyses with their low proportion of measurement studies. The EMF studies with full-shift measurements provide more reliable data on cancer, both because the measurements give a consistent exposure

metric across studies, as well as having better epidemiologic designs and data. The primary shortcoming of the comparative analysis is its omission of 6 cancer studies with full shift measurements – 2 studies in electric utilities and 4 with subjects employed in other industries. These other studies provide a mixture of positive and negative results, and the NIEHS working group report (1998) found their designs generally weaker than the four studies in Kheifets 1999 paper.

The exposure-response relationship in the Khiefets analysis also makes it possible do a cost-benefit analysis for occupational EMF expenditures. This methodology is a strong part of the California report, and could help in the analysis of occupational EMF policies.

In general, these two reports promise to advance the evaluation of EMF risks and the analysis of policy options. Addressing the issues raised above would help these reports achieve their potential impact.

Specific Comments on
the EMF risk evaluation (chapts. 1-9) and policy analysis
by the California EMF Program

Page #	Table #	Line # or table footnote #	Comments
10	1	Prior confidence	The values on this graph differ from the narrative in chapter 2, especially with reviewer #3.
17		25	Needs units (deaths per year, etc.)
31 (also A-30)	3.1.1	Also, 24-31	The discussion of the EMF mixture does not recognize that the fields are time-varying vectors. What the text calls "magnetic field intensity" or "the field" or "a measurement" is actually the rms vector magnitude over a ~0.1 sec period (AKA the resultant). NO proposed biological mechanism says that the ELF magnetic field magnitude is the causal metric by itself. Other vector properties such as the polarization and the rms component of the ELF vector perpendicular to the static field vector are important to biological effects, according to some proposed mechanisms (see Bowman and Methner, 2000).
31		34-35	The citation to Neutra, 2001 is missing from the references.
32	3.1.1		There's a typo here where X comes out as
32	3.1.1	Row (7)	Burch et al. (2000) report melatonin changes among electric utility workers in 3-phase environments, which they attribute to, the polarization differences between 1- and 3-phase sources.
32	3.1.1	Row (10)	Associations of rate of change metrics with melatonin are reported by Burch et al. (1998; 1999)
32		13-21	Should give values for the correlation of the TWA with other metrics, plus references. The rms vector magnitude also has strong to "modest" correlations with other ELF magnetic field characteristics (Bowman and Methner, 2000).
73		1-5	As discussed in the text, the comparative analysis by Khiefets et al. (1999) should be used for adult leukemia in addition to the 1997 meta-analysis.
89	8.2.2	Item (A2)	In the comparative analysis, the pooled OR = 1.48 (0.96-2.30) for adult leukemia in the highest exposure category. This is less likely to be due to bias than RR=1.2 from the meta-analysis.

Note: Highlighted words are suggested insertions. ~~Strikeouts~~ are suggested deletions.

89	8.2.2	Item (A5)	The studies in the comparative analysis all use state-of-the-art methods for occupational cancer cohort studies. The cohort method greatly reduces selection and information bias. The significant association from these high-quality studies are not likely to be due to bias, making them evidence for causality.
92	8.2.4	Item (F2)	Exposure assessments in occupational cancer studies are not "inevitably poor". That is true for many of the studies in the meta-analysis which assess exposures with job titles, but the studies with measurements used a job-exposure matrix, which is the state-of-the-art method for occupational epi and has detected many workplace carcinogens. JEMs do have exposure assessment errors from linking measurements by job titles, but residential studies have errors too (e.g. using an area measurement to represent personal exposures). Comparing the effects of these errors on risk estimates is very difficult, especially because residential studies generally have classical measurement errors (biased towards the null) and JEMs have Berkson error, whose direction of bias cannot be predicted from first principles.
93	8.2.5	Item (C1)	"Lack of statistical significance is not only related to the likelihood of causality, but also to the study power."
93-94	8.2.5 and 8.2.6		Since the combined analyses of the residential studies (Greenland et al; Wartenburg) and the electric utility studies (Kheifets et al., 1999) with measurements all find the data to be highly homogenous, the seemingly inconsistent pattern of statistically significant risks is apparently due to chance fluctuations. Since the TWA magnetic field magnitudes are roughly correlated with the true dose metric, such fluctuations among studies is to be expected. This goes in the "For Causality" column.
94	8.2.6	Item (A3)	This argument is totally unclear. More details are needed as to what studies are homogeneous, and which are not (plus references). Also, the logic why this is evidence against causality is lacking. P.S. This item should be (A2).
94	8.2.6	Item (F1)	The distinction between the Greenland studies which "were homogeneous" and those in Kheifets which "were not heterogeneous" is unclear.
94	8.2.6	Item (C1)	This studies with wire codes are not homogeneous.

94	8.2.6		Wire codes are the poorest of all surrogate exposure measures. The demise of the wire code paradox has greatly diminished the hypothesis that wire codes capture a key exposure characteristic that is not being measured. So any argument against causality on the basis of wire code evidence is completely trumped by the results from measured and calculated exposures.
95	8.2.7	Item (F1)	"All studies use surrogates exposure measures."
97	8.2.8	Item (C3)	"… is not a big concern" would be better stated as "….does not change the likelihood much in the final evaluation."
102		27-30	This sentence is not true for all types of occupational studies. First, the occupational EMF environment in general industry is more heterogeneous than in homes (Bowman and Methner, 2000). In electric utilities, the magnetic fields probably have less harmonics, but the electric field and magnetic field polarization are probably more complex. Second, the amount of bias (recall and otherwise) depends on the study design. Cohort studies that rely on corporate job records have no recall bias. The studies in the Kheifets comparative analysis have less recall and selection bias than the childhood studies.
110-114	9.1.2		This chapter lacks any textual argument to justify inclusion of the range of study designs in the Kheifets meta-analysis. The analysis seems to give no weight to the observation that the more modern, better conducted studies show no increase in brain cancer odds ratios
112-114	9.1.2		The inclusion of 3 early studies (Speers 1988, mean odds ratio 3.94; Thomas, Stolley et. al.1987, mean odds ratio 2.30; Coggon 1986, mean odds radio 2.0) with their high odds ratios may skew the overall results.
118	9.2.4	Item (A1)	A study with a risk ratio that is that is not far enough removed from 1/1 to be statistically significant is not proof of causation and does not become probative when combined with other studies with the same flaw.
Policy, p. 2		57	Name and/or reference the "policy contractors".
Policy, p. 3		19-20	The cost benefit analysis is based on a theoretical $5 million per life saved hypothesis. This hypothesis could benefit from some foundation.

Policy, p. 3-4			This analysis should examine if the populace is as willing to spend $5 million on 10% certainty as they are on a 100% certainty. The utilitarian analysis needs to be factored for degrees of certainty.
Policy, P.4		10-12	Inserting a plastic segment in water pipes does not eliminate all household fields, and would appear to be cost-effective only after a diagnosis of field sources in the home. See Leeper (2001).
Policy, p. 6		20-23	The calculations may be more meaningful if the dose response was used to calculate the cost per milligauss-hours avoided. This approach is suggested here, but does not seem to be completed.

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January 11, 2002

Dr. Raymond R. Neutra
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1515 Clay Street, Suite 1701
Oakland, CA 94612

Dear Dr. Neutra:

Following up on our discussions at the meeting of January 9, 2002, I am providing answers to your pre-meeting questions.

1) [**Re your overall response to comments**] I think most of the comments received, pro and con, reflect more about the commenter's comfort with the conclusions than about new insights on the methods of analysis. Overall, I think you and your staff did a good job of summarizing the major points of criticism, both those answering your 8 specific questions and otherwise. I was somewhat less pleased with your responses to criticisms and your corresponding proposals for modifying the report. For example, some of your replies to the comments of Sander Greenland seemed unreceptive. Although I admit that my understanding of the statistical issues raised is limited, your responses did not always make more sense to me than Greenland's arguments. However, I do not think that his criticism regarding qualitative vs. quantitative Bayesian analysis is fully justified. I think he will find plenty of criticism regarding which data he chooses not to use in the quantitative analysis and how he assigns relative likelihoods to each data set he does use.

After answering your other questions I will return to this one with some examples of points that may deserve more effort on your part.

2) [**Re new probability categories and English phrases**] The discussion at the meeting was very useful. I think Tom McKone's suggestion to use statements about the extent of your belief in the existence of a hazard is good, although you must still exert care to distinguish likelihood of hazard from degree of risk. I agree that the language regarding what future research will show is ambiguous and should be struck. Note that such language must also be struck throughout the statement to the public. I am not much concerned about the numeric cutpoints for the plain language statements, although having too many or two few categories is less desirable. I suspect that five categories may be enough.

3) [**Re statement to the public**] Except for the language on future research, I wasn't unhappy with the new statement, although I agree that it is difficult to know how it will be read. There are a few other tripping points:

p. 14, first paragraph, line 4, after "population": maybe "lead them toward the belief that EMFs increase the risk of some health effects . . ."

p. 14, second paragraph, line 1, after "reviewers": maybe "express the degrees of their beliefs as numeric probabilities. For . . . probabilities they believed supportable, as follows:"

Why is spontaneous abortion in a separate table from the other effects? Also, shouldn't you be consistent in calling it spontaneous abortion or miscarriage?

p. 17, second paragraph, lines 1-2: “might cause a substantial proportion of miscarriages” seems a stronger statement than elsewhere, where you talk about “added risk”or use other wording. The miscarriage wording sounds like strict causation rather than contribution.

P. 17, last paragraph, line 4: “real culprits” may reveal a bias towards the need to find blame. I suggest something more neutral.

4) [**Re expansion of executive summary**] Any solution will have its virtues and faults. I agree that the detailed technical treatment should not be described as an appendix, or even an annex. Maybe there should be several separately bound products so that any one reader could acquire only the level of detail desired, e.g., Statement to the Public (which would be close to what is needed for a press release), Executive Summary (maybe five pages and directed toward decision-makers), Technical Summary (maybe 30-40 pages, more or less what you suggested in this question), and Detailed Technical Report. I agree that you must be extremely cautious in creating any of the shorter documents to avoid ambiguous wording, over- or understatements, etc.

5) [**Re further recommendations**] None not covered elsewhere.

Detailed response to Question 1):

Lack of mechanism (see, for example, Dawsey p. 7): This gap continues to drive my own beliefs down. Although I agree that absence of evidence isn’t evidence of absence, I think you are too dismissive (e.g., your p. 11, item 13) of the lack of success in finding a plausible mechanism and verifying its presence and place too much reliance on analogies with agents with much stronger epidemiology. I don’t expect you to change your numbers, but you might more strongly acknowledge the alternative view.

Alleged mistakes of logic (Greenland p. 4): These accusations (misinterpreting evidence, double counting, ignoring flaws in epidemiology) may or may not be accurate, but I didn’t find your responses particularly thorough or convincing. The double counting issue was addressed in your Item 7 on p. 7, but it made a counter allegation against Greenland that I did not find to be on point. It was also addressed in I I’d also like to understand better Greenland’s claim that tem 9 on page 8, but I didn’t find the response convincing. When most of the epi studies are not statistically significant, should you be counting both the proportion of studies with OR>1 AND some subjective evaluation of how closely the ORs resemble one another? Did you adjust the ORs for calculated or measured exposure in each study?

Alleged misuse of sign test (Greenland, Kavet): You responded in Item 8 on pp. 7-8 and in Appendix 4, basically to the effect that the sign test evaluates what the sign test measures. Greenland seems to think that it doesn’t take into account the possibility that shared biases, confounders, etc. affected the pattern. I don’t have the statistical and epidemiological expertise to support Greenland, but your responses weren’t sufficient to dismiss him, either.

Selection bias (Greenland, p. 23): You responded at length in Item 12, pp. 9-11. Again, I don’t have the expertise to evaluate this issue, but I saw some tendency for you to play up the arguments against selection bias and play down those for it. The opposite is probably true for Greenland. This illustrates my point that it is the rare expert who can be utterly objective in the presence of his or her own previous experience and worldview. Again I would not ask you to change your evaluation, but I do think that some discussion of reviewer choice (Item 3, p. 5), needs to be included in the final report, with appropriate condensation in the various summaries. Especially important is the middle paragraph of the right column on p. 5.

Claim regarding confounders (Greenland, p. 27): Greenland accuses you of a vast overstatement regarding controlling for confounders, and I think I agree. I suggest you weaken the claim. Also, you defend your discounting of the effects of confounding by claiming (detailed comments, p. 45) that breast cancer and ALS are not correlated with benzene. Do you have citations for studies looking for a benzene connection, or are you, now, the ones confusing absence of evidence with evidence of absence?

Saturation (plateau) in the exposure-response relationship (Greenland, p. 31): Greenland make a probably unjustified ad-hominem attack regarding post-hoc explanations, but the point is still one that bothers me: why should we believe in this rather unusual pattern?

Selective reporting of dose metrics (Jaffa, p. 4): The claim was made here and at the meeting that CDHS did not report all of the results when more than one metric was used for analysis of childhood leukemia epidemiology. The implication was that CDHS selectively reported the ones that produced ORs greater than one. While it is true that the study would be "positive" if any metric produced a statistically significant result, this practice would probably inflate the apparent consistency of the studies. I don't know whether this allegation is true, but it should be checked and, if so, appropriately corrected.

Laws of nature vs. models (Sahl, p. 4): Sahl correctly distinguishes between "laws," which are widely assumed to be absolute and that have been subject to multiple independent tests, and less global theories, which you have called "models" and which represent substantially less confidence. You basically agree, and the debate is then about whether the claim that EMF energies are insufficient to supply a biological signal is in the former category or the latter. Although I tend to agree that strict violation of, say, the second law of thermodynamics is not required for EMF effects to be true, I also think that one needs both a biological antenna AND a biological amplifier to raise those energies to signal level. The existence of phosphines, magnetite nodules, etc. provide some plausibility for an antenna, but I don't think bioamplification of aural signals (Adey) or photons of red light (your detailed responses, p. 5) are strong arguments for the existence of an EMF detector. I'm not even sure they are true: do you have citations? Again, absence of evidence is not evidence of absence, but those who doubt the EMF hypothesis would more easily convinced if an EMF antenna and amplifier were identified. What should you do about it? Just be even handed in acknowledging the problem.

Cutoff date for literature (e.g., p. 10 of your detailed responses): Some confusion remains in my mind about your application of the June 2000 cutoff. If it really required publication by that date, then the two CDHS miscarriage studies would not qualify. Although I am not personally bothered by your use of them per se, there are questions of evenhandedness if you have ignored other publications since that date. You tried to explain the rule better at the meeting, but I did not fully understand. Please be sure that the explanation is clear and no significant bias is involved.

Anchor for prior (p. 17 of your detailed responses): I continue to maintain that anchoring with a supposed prior for chemicals (I think specifically, chemical carcinogens) is not justified. Chemical toxicity, especially carcinogenicity, has convincing evidence for causal mechanisms. EMF toxicity does not. Would you apply the same prior to the hypotheses that listening to symphony music, viewing abstract art, or indulging in political discourse are hazardous to specific health endpoints, just because they are manmade? Surely those are inappropriate comparisons, but they argue against a firm anchor from the chemical realm. I would go further and say that one's priors for different endpoints for EMF toxicity could--and probably should--be different.

Statistical significance (re Appendix 4, pp. 9-10): Although I thoroughly agree that statistical significance is an arbitrary test that should not completely discount suggestive evidence, the table on these pages makes the evidence for childhood leukemia seem a bit stronger than it really is. If I recall correctly, most of these studies did not reach statistical significance as reported. Maybe a column for significance should be added.

Socioeconomic status as possible confounder (Appendix 5, p. 12): I found this explanation almost totally confusing, especially the tables. Does the second table mean that the association goes away (OR<1) if SES is included in the analysis? If not, what does it mean?

Comparison of CDHS approach with that of expert panels (Item 2 of your responses, p. 5): I support the idea that your approach is not *a-priori* worse than that of a panel, and probably is better because the judgements are made more explicit. But it is probably unnecessarily pejorative to say (line 2) that evidence is only "briefly" discussed by the experts. Suggest deleting the adjective.

Sincerely,

Stephen L. Brown
Director

Box 0808, Room N431
UCSF Cancer Center
University of California,
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August 13 2001

Dear Dr Neutra,

I wish to address the conclusions of the EMF panel, especially with reference to the discounting of prior biophysical considerations from the weight of evidence. To illustrate my response I am attaching a sketch of the electromagnetic spectrum, upon which I have marked the wavelengths and energies corresponding to power lines (6 Hz) and cell phones (approx 1000 Hz) (Figure 1). This is important, in order to depict residential EMFs in the context of sound biophysical understanding of th electromagnetic spectrum and its range of interactions with matter.

DRAFT REPORT - REJECTION OF BIOPHYSICAL THEORY

The Draft Report 3 claims that "...the established physical theory and simplified biological models, together, were not sufficiently strong to prove the impossibility of epidemiological or laboratory observations" (21.2.1). A claim was also made that "...as was also the case for many disease-causing agents, there is not a well-documented mechanistic chain of events that explains how the EMF "mixture" at residential or occupational levels could initiate a biological response..... (21.2.2).

I question the perspective of this opinion, the interpretation of current biophysical theory, and the use of analogies to paradigm shifts in scientific understanding. Novel interpretations such as Wegener's continental drift represents initial observations about which there was much controversy. With the passage of time and the accumulation of additional data the evidence strengthened and now has entered general acceptance in the scientific community. The possibility of EMF health effects came on the scene with Wertheimer and Leeper's original analysis 20 years ago which was based on leukemia incidence correlated with weak and poorly quantified EMF exposure estimates. The subsequent 20 years of investigation has not improved or strengthened the purported correlation, and our present biophysical understanding would support the view that there is nothing to explain.

INTERACTIONS WITH MATTER

In order for any agent, radiation or chemical, to be a human or animal carcinogen the agent must have the ability to interact with cellular systems and macromolecules. Upon absorption there must be enough energy to disrupt chemical bonds and initiate a destabilizing effect on the genome. This is clearly understood in general outline for ionizing radiation, ultraviolet light and many electrophilic chemical carcinogens. Very few agents that are carcingenic in humans are without demonstrable effects in bioassays. EMFs at power line frequencies, however, do not have the necessary energy to interact with biological molecules (see Figure 1), and do not give consistent results in standard carcingenicity assays.

The interaction of EMFs across the whole spectrum is sufficiently well understood that we can confidently state that the low frequencies associated with power lines and cell phones will not have adverse health effects. Any epidemiological suggestions of an apparent association between power lines and adverse health effects should therefore be a stimulus for searching for other confounding or different causal effects which are not due to the electric and magnetic fields.

The absence of a causal chain of evidence for some other agents (e.g. aspirin) is only an absence of detail within general knowledge that there is a chemical and biochemical process. For EMFs in the power line frequency range there is not just an absence of detail, but total absence of an effect to explain, for sound

physical reasons. We have a positive understanding that the power line frequencies do not allow sufficient molecular interactions to have any biological effect.

THE ELECTROMAGNETIC SPECTRUM

EMFs associated with power lines represent one extreme of a wide spectrum over which the wavelengths and quantum energies vary by a factor of 1016 (Figure 1). Thus the energy associated with a quantum of power line EMFs is nearly a billion billion times less than gamma and Xrays.

At the short wavelength extreme of the spectrum, gamma and Xrays have undoubted biological effects. This is because the quantum energy of the radiation is high enough to ionize atoms, create free radicals and initiate a chain of radiochemical reactions that can cause major disruption of genetic stability. At somewhat longer wavelengths, associated with ultraviolet light, molecular changes are caused by absorption in double bonds of pyrin-tidines in DNA and lead to the well-recognized role of solar UV light in skin carcinogenesis. Thus the major carcinogenic range for electromagnetic radiation is on the short wavelength side of the visible spectrum (Figure 1). Fundamental to these deleterious biological effects is the important principle established in radiobiology, that only absorbed energy can cause biological effects. Absorption is possible because the quantum energy of the radiation is sufficient to ionize and excite biological molecules. There are "receptors" for the radiation in the electronic and molecular structure of biological molecules. Therefore these deleterious effects that are indisputably caused by absorbed energy of short wave length electromagnetic radiation represent the well-understood biophysical underpinnings of radiation carcinogenesis.

On the long wavelength side of the electromagnetic spectrum the situation is quite different because of the lower energies involved. This longer wavelength extreme of the EMF spectrum is beyond the range for which there is sufficient quantum energy in radio and power line frequencies for absorption in any biological molecules. At this extreme the receptors are long metal wires or metal dishes, such that only free electrons in the energy levels of metallic materials can absorb sufficient EMF energy to detect and respond to these EMF frequencies. Note, that the important factor here is not the strength or intensity of the EMFS, but the fact that the frequencies are so low, and wavelengths so long that the quantum energy is too low to interact significantly with biological molecules.

We are therefore talking here not about the absence of theory, but the presence of a well-founded theory for EMF interactions with matter. On this basis, the weight of evidence is that EMFs in the cell phone or power line frequency range do not interact sufficiently with biological material to initiate or promote cancers. Epidemiological data 91 that imply an association between EMF exposure and cancers should therefore be used to search for other confounding causes than the magnetic or electric fields.

DOSE REPONSE RELATIONSHIPS

The statement is made that these criticisms from a biophysical perspective require demonstration of "...robust bioeffect laboratory results from ambient levels of exposure." (section 21.1 lines 12-13), and that this represents a higher standard than required for other epidemiological agents. I am unaware that we have made this a requirement anywhere in the analysis of biophysical interpretations of EMFs. What is required is only the same standard should be used for hazard assessment as is used for Xrays and UV light, that are shorter wavelength electromagnetic radiation. This requirement is that over a range of intensities of 60 Hz power line frequency Ews, using an established assay for which there are both positive and negative controls (e.g. chromosome aberrations, micronucleus, transformation, animal carcinogenicity), there should be a dose-response relationship for a biological effect associated with various exposure levels of 60 Hz power line frequencies. A dose-response relationship then allows, as for Xrays, extrapolation to ambient levels and a quantitative assessment of health risks for cancer or other end points. Thus far, these basic criteria, that all putative carcinogens must meet to be classified as a human hazard, have been negative or inconsistent for EMFs at power line frequencies at any intensity. I am therefore confident that we have an adequate biophysical understanding of how EMFs interact with matter and this is consistent with the absence of positive results in short-term laboratory experiments and animal carcinogenicity assays.

I hope that these opinions can be taken under consideration, because I believe that the report dismisses a substantial body of scientific knowledge, and gives an incorrect emphasis to interpretations of epidemiological data.

Yours sincerely,

James E. Cleaver PhD

NOTE: These opinions are my own, and in no way are intended to reflect any official position of my employer, the Universty of California, or any other agency for which I consult or serve in an advisory capacity or other panels on which I serve.

From: Roger Conant [mailto:rconant@powerlinefacts.com]
Sent: Monday, September 10, 2001 2:28 PM
To: emf@dnai.com
Subject: Comments on The Risk Evaluation and the Policy Options Summary

Dear California EMF Program:

You invited comments on The Risk Evaluation and the Policy Options Summary.

We have only one comment.

Throughout the report, you refer to the likelihood of an event occurring as a "possibility," even when the likelihood exceeds 50%. For example, on page 3 of the Scientific Abstract, you refer on line 6 to a "more than 50% possible that residential or occupational EMFs could cause childhood leukemia, adult brain cancer, ALS (Lou Gehrig's disease), and miscarriage."

With reference to likelihoods greater than 50%, this use of the word is not consistent with common usage in ordinary language nor is it consistent with normal statistical nomenclature at any degree of likelihood.

Instead, the word "probability" should certainly be used in connection with all likelihoods greater than 50%, and arguably would best be used for all likelihoods.

Accordingly, the above sentence would be better articulated as: ..."more than 50% probability that residential...."

Thank you for considering this comment.

Sincerely,

Roger R. Conant, MGA, Ph.D., CLU
President
Power Line Task Force
2 Sunfish Lane
Sunfish Lake, MN 55118

August 6, 2001

To: Dr. Warren Winkelstein

From: Dr. Carl Cranor, carl@chss.ucr.edu

Re: Comments for EMF Science Advisory Panel Meeting

I will not be able to attend the meeting in Oakland on Tuesday, August 7, 2001. By this email message I request that you read my relatively short remarks to the panel. At about 2:15 California time, I will join the open discussion by telephone from the Denver airport.

In general, I have found this risk assessment document much better, more transparent, more informative than almost any risk assessment document that I have read in my research or while serving on other science advisory panels. What goes into this is the following. I have a reasonable understanding after assimilating it why the different scientists involved reasoned and concluded as they did. The process of making the sausage is quite clear, which has both good and bad aspects to it. It is desirable because one can tell why each person reached the conclusions he or she did and why and how this compares with other evidence evaluation decisions. It also makes it easy for critics because they will be able to focus their comments quite precisely at a particular step in the reasoning process. Nonetheless, the transparency is to be applauded, especially in a controversial area such as this. If this document passes muster with the peer review panel, despite what will likely be criticisms of it (not because there is anything wrong with the document or the process, but because it comes to conclusions that some interest groups will object to), it should help educate, inform and persuade the public of its conclusions. Most important, the broad public should not feel, as they would with many documents, that there is a lot of mystery here or that there is a lot that is hidden. Ultimately, it should result in greater public support for the assessment.

In particular, I found the summary comments to the public as helpful as they are likely to be. Since they are hedged in various ways in order to be accurate and not misleading, that hedging may to some extent be slightly confusing. However, that is unavoidable confusion, given the complex, subtle technical issue involved.

The comparisons with IARC and with other environmental hazards were helpful in providing perspective. The discussion with the scientifically quite conservative IARC was helpful for providing context. It is important to realize how reluctant IARC is to draw conclusions that a substance has an adverse health effect (because of very high evidence demands) and to compare California's approach, which is less likely to result in false negatives (FNs), a concern quite important to the general public. An agency should attend both to the possibility of FPs and FNs resulting from an assessment of evidence. There may be contexts in which one can afford to focus more on avoiding one mistake than another, but the importance of attending to both in this context cannot be overemphasized. It is especially important for an agency to be aware of a stream of evidence that "is prone to miss picking up disease-causing agents". Finally, in my view agencies charged with protecting the health of the public should not be so extremely cautious about avoiding FPs that they incur the equivalent of regulatory FNs through inaction or unreasonable demands for evidence.

The document's idea of streams or patterns of evidence is quite valuable. Articulating the relationship between different "streams of evidence" and the reasons why one is poor, inadequate, or supersedes another is valuable for its own sake, for theories of evidence evaluation and for providing scientific and public support for this document. I thought the document puts mechanistic evidence into a proper perspective for purposes of public policy issues and what is usually available given great scientific ignorance about many environmental toxicants: even for well-studied substances such as aspirin neither the mechanisms of beneficial nor harmful effects tend to be known in any detail (according to standard textbooks on the issue).

The discussion of the prior and its basis, as well as discussion of the kinds of evidence that motivated the reviewer to change the prior is far superior to what one finds in other risk assessment documents. It is important

to note the role that hidden differences in the prior can play in evidence assessment (Introduction, p. 4:26) as well as where differences in the weighting of evidence occur.

Creating pro and con argument tables concerning each of the diseases in question and assessing the evidence by means of Hill's considerations is quite helpful to non-experts and I would think to the public. Transparent although this is, it will probably still be difficult for the broader public to understand, but is probably about as good as one can do in the circumstances.

I continue to have a disagreement concerning the categories of confidence whether or not EMFs make a causal contribution to adverse health effects (9.3-18 and in Introduction). The category--possible greater than 50% confidence--should have a different word. It seems to me that this terminology is simply misleading. Why not use the phrase "probable, greater than 50% confidence." The reason I suggest this usage is that it conforms more to ordinary uses of the term "probable". Moreover, how we use words makes a difference. I am concerned that some in the public will seize on the word "possible" to claim that this shows, despite the rest of the phrase, that it is only a possible adverse effect, not a probable one. Moreover, the suggested change fits better with the other probability words that are used in the document.

Finally, a few comments on the Policy Options document. This paper nicely complements the scientific analysis. While I find some of the philosophic positions providing the foundations of different reactions to the scientific analysis a bit overly simple, they do provide foils for how to think about a proper social policy for EMFs if they have real adverse effects. More important, the typical approach to risk management never considers the different foundations for policy that are made explicit here. Consequently, in most risk management decisions these considerations are hidden or addressed from only one framework, usually some kind of cost-benefit analysis point of view. This last observation highlights one more way in which the EMF risk assessment and risk management exercise is more comprehensive, transparent and informative than most one reads. I found the Policy Options document quite helpful, one that is also likely to be quite helpful to the public as well. One addition I would suggest (at least given the copy I had available) is to make explicit in the abstract for the public the conclusions of the document itself. Those conclusions were not in the paper I read.

I support the procedure used by the HHS scientists and the conclusions at which they have arrived. Please cast my vote in support of the procedures, conclusions and the document. That others might disagree with some of the assessments is only to be expected for such complex, subtle, difficult issues. If there is a need to vote on some other motion than an overall assessment of this document, perhaps I can provide that by telephone on Tuesday at 2:15-3:30. If you need anything further from prior to the meeting I will be in my office most of Monday, August 6 (Phone: 909-787-2353).

Shan Cretin
RAND Health Program
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Answers to questions:

1. I agree that the lack of biophysical models currently able to explain an effect should not create a prior so strong that epidemiological data could not possibly affect the posterior. However, I do think that marginal or equivocal epidemiological data will have stronger weight in the presence of a well-understood mechanism. Is this the same thing as saying that the lack of a theory leads me to discount evidence? I think so, in that the conditional probability of there being a real EMF affect given the same set of evidence AND a plausible mechanism is bigger than the conditional probability of there being an effect given the same set of evidence and NO plausible mechanism.

2. This is not my area of expertise, but I think I would have had relatively lower a priori estimates for EMFs. I am influenced in this by the lack of biophysical models and also by the sense that EMFs are likely to be correlated with a number of other factors that could be the culprit even if there are real and enduring associations between EMFs and certain cancers.

3. Perhaps I have already addressed this in Question 1. If there was a convincing mechanism, then the epidemiological studies should be more convincing and more interpretable. For example, if we know exposure is related to the cumulative dose over time or the presence of EMF lowering resistance to other carcinogens, then the epi studies could be interpreted in light of the appropriateness of the exposure measures and covariates. Without a mechanism to fall back on, it is harder to know what missing measures might explain away apparent effects or how poorly chosen exposure measures might fail to find true effects. So the lack of plausible causal mechanisms undermines my faith that the epi studies are appropriately designed, making me more apt to treat them as noise.

4. The lack of animal effects does pull down my confidence in most human effects for reasons similar to those stated in 1 and 3 above. However, this would be a relatively small effect, since some diseases don't have easy to find animal models. The chick embryo studies *only* have *increased* my confidence for the possibility of effects on fetuses and very young children.

5. Relative risks between 1 and 2 are not terribly convincing to me, especially when those risks are closer to 1 than to 2. I view the absence of a plausible explanation for confounding/bias as similar to the absence of a plausible biophysical mechanism. Surely there could be sources of confounding/bias not yet identified! Your assessment of these equivocal risks says that you are taking a conservative stance, in the sense of being biased towards finding an effect. As public health policy makers, this may not be an unreasonable approach but it is a bias.

6. The lack of specificity with regard to subtypes of cancers does weaken my view of the evidence. I'd certainly be more confident of an effect if faced with consistent, specific disease-exposure associations. I guess the main support for increasing confidence is if the posited mechanism is that EMFs somehow increase the "infectivity" of other agents. However, I was surprised by your argument here and do not agree.

7. While you did a reasonable job for many of balancing pro and con arguments, I sometimes felt that the pro arguments were presented more strongly than they deserved. For example, there are underlying assumptions that studies are independent, but in fact, the study designs, measures and choice of covariates are quite similar across studies so that unrecognized flaws may (probably do) propagate through the literature. In general, scientists tend to think they know more than they do from individual studies. Physical constants measured in early experiments tend to share biases so that the ultimate number agreed on for the constant is often found to be outside the confidence intervals of the early studies.

8. One problem with this table is that I would not call 2% "virtually certain"—I'd save that for a 995 in a thousand (or 5 in a thousand) chance. Another is that I would have a neutral mid band around 50. Words mean different things to different people, however, which is why we use numbers! My words would be: 99.5% virtually certain; 99.5 to 98% highly probable, 90-98% probable; 60 to 90 more likely to be causal than not; 40 to 60 equivocal; 10 to 40 less likely to be causal than not; 2 to 10 improbable; 0.5 to 2 highly improbable, 0.5 virtually certain not causal.

Another way to approach putting words to numbers is to reinterpret the numbers to everyday events: The risk of being in an auto accident if a 30 year old drives X miles. (putting 30 year old in to take care of the fact that 16 year old boys or 87 year old women might have a different experience). Or the probability that there will be a 6,0 or larger earthquake in California in the next N minutes (hours, days?)

Other comments:

I continue to be concerned that the model does not do an adequate job of clarifying what the *independent* contributions to changes in property value might be, besides health effects, aesthetic effects, pole crashes and other items already included separately. While health effects and aesthetics and pole crashes have impacts in and of themselves, I don't think the property value changes measure an impact in and of itself. I see property value depreciation is a market driven surrogate for the sum total of all effects that a transmission line has on a property of which health effects are one component.

The (probably unanswerable) question is this: how much is property value a reflection of (a) the expected health effects you are calculating and how much is it a reflection of (b) other things (irrational fears or aesthetic factors not captured elsewhere in the model)? To the extent that the property value impact is due to (a), then double counting is occurring. To the extent property value changes reflect things NOT captured elsewhere, then it is appropriate to include them. I would like a clear statement that the calculations including property value effects are overestimates. The difficulty is that we don't have any idea how large an overestimate based on the report.

Unfortunately the property value impacts in the models are large, while the impacts of esthetics, pole crashes etc are small. It seems likely that perceived health effects are a large part of the property value change (with the caveat that the perceived health effects may be quite different from the effects being estimated based on the literature).

On a different topic, I am not certain that the social justice perspective is a fundamentally different framework. It seems to me that the social justice perspective be captured by adjusting utilities in the decision tree, reflecting the fact that given the same decision tree, different stakeholders with different utilities for certain outcomes will come to different conclusions. So if equity is part of the concern for a stakeholder, then the set of outcomes on the decision tree can reflect equity as yet another attribute to be assessed. There needs to be a clearer distinction between different frameworks and different interests

September 9, 2001

Jack Collins
California EMF Program
1515 Clay Street, Suite 1701
Oakland, California 94612

Dear Mr. Collins:

Following are comments on the draft California Department of Health Services (CDHS) EMF Report: "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances," April 2001. The comments focus on the review of the Report's findings and conclusions regarding spontaneous abortions.

Critique of the General Approach to the Evaluation of Health Risks from EMF's:

The general approach taken by the DHS to evaluate health risks from EMF's is very sound and well reasoned. Findings are summarized in two ways: 1) the degree of confidence that EMF's cause disease is stated according to a set of Risk Evaluation guidelines developed by DHS for this purpose, and 2) evidence is classified according to the traditional IARC guidelines. The report clearly spells out how each reviewer applied the DHS Risk Evaluation guidelines and IARC guidelines to arrive at the degree of confidence and IARC classification for each disease endpoint. The presentation of pro and con arguments according to a series of pre-specified questions relevant to causality (i.e., chance, bias, confounding, strength of association, consistency, homogeneity, dose response, coherence/visibility, experimental evidence, plausibility, analogy, temporality, specificity, and other disease associations), dose response, and policy makes the thought process of the reviewers explicit and clearly delineates how the conclusions were arrived at.

Critique of the Conclusions Regarding Spontaneous Abortions:

Statement to the Public: This statement on page 186 clearly summarizes the reviewer's conclusions regarding the risk of spontaneous abortion from exposure to EMF's according to the two sets of guidelines. While the designation of '*possible risk'* according to IARC guidelines, and the statement '*50-90% degree of confidence in causality'* according to DHS guidelines follow from the detailed presentation of pro and con arguments, it is not clear how the statement regarding the *addition of 5-10% to the baseline risk of miscarriage* was derived. It would be helpful if the report included a description of how the figure of 5%-10% additional risk was derived from the available data presented.

Evaluation of VDT Studies: In the summary statement of Reviewer 1 on page 206, an argument is made that the results from the VDT studies can only be used as a
strengthening-only type of evidence because exposures from VDT's are substantially lower than the residential and occupational EMF exposures that are the focus of the risk evaluation. It would be helpful to the reader if a similar statement regarding the low level of EMF's emitted from VDT's was included in the summary statement about the VDT studies on page 188.

Evaluation of Personal Measurement Studies: Overall, the findings from the two personal exposure measurement studies by Lee et al (2000), and Li et al (2000) provide the most compelling evidence for a possible positive association between EMF exposure and spontaneous abortion. The maximum field metric was found in both studies to be associated with an approximate 2-fold risk for spontaneous abortion when the field exceeded 16 milligauss. However, there were a number of limitations of each study, and inconsistency of results between the two studies, which need to be addressed before the degree of confidence in causality could be increased such that the probability of no effect would not be included in the statement of causality. The most important limitations of both studies are the low participation rates and retrospective assessment of exposure. Both limitations may have introduced a selection bias resulting in differential misclassification of exposure, which could bias results away from the null value. Furthermore, the Lee study reported a dose-response effect of maximum field exposures but the Li study found a plateau effect. Finally, neither study provided information about the sources of the brief, high field exposures, but suggested that these exposures could come from electric appliances or other electric devices inside and outside the home.

Evaluation of the Conclusions: The DHS reviewers conclude that the added risk EMF poses on miscarriage is of regulatory concern if the associations found with the change in magnetic fields and brief high fields metrics are true. They suggest that "policy should focus on developing strategies to reduce or avoid these types of fields, which will require policy to direct funding for future studies to understand the nature of the exposure, to evaluate the sources of such fields, and to explore methods for mitigation." This reviewer is in full agreement with the conclusions of the DHS reviewers. The immediate next step is to provide funding for research that will attempt to characterize the sources of the brief high fields and changes in fields that are experienced by the majority of women during their pregnancies. This type of research is necessary in order to formulate rational policies that address the mitigation of potentially damaging EMF exposures.

The views expressed in this document are solely the author's and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas & Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District, and San Diego Gas & Electric Company.

Sincerely,

Lisa A Croen, PhD

September 10, 2001

Dr. Raymond Neutra
Division of Environmental and Occupational Disease Control
California Department of Health Services
1515 Clay Street, Suite 1701
Oakland, California 94612

Dear Dr. Neutra:

The California Department of Health Services (CDHS) has requested comments on their *draft* EMF Report: "An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances" (*draft* 3, April 2001) and the Draft "Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields (EMF)" (collectively "Draft"). The Draft was prepared at the request of the California Public Utilities Commission (CPUC) in its Decision No. 93-11-013 (November 2, 1993) and may serve as the basis for the CPUC's further consideration and implementation of EMF policy within California.

These comments are submitted jointly on behalf of the following utilities: Los Angeles Department of Water and Power; Pacific Gas and Electric; Modesto Irrigation District; PacifiCorp; Sacramento Municipal Utility District; San Diego Gas and Electric; Sierra Pacific Power Company; and Southern California Edison. Collectively, these utilities serve over 20 million customers in California with electricity.

We appreciate the hard work and long hours CDHS staff have given to this important project. Our comments on the Draft will be made in three different ways. First, in this letter, we provide summary comments to focus CDHS's approach towards appropriate revisions to the Draft. We also provide recommendations on how to improve the CDHS process to revise and finalize the report. Second, we have funded ten independent experts who have reviewed the Draft and who will submit their comments on specific sections of the Draft directly to CDHS. Third, we are members of organizations that will also be submitting comments (e.g., EPRI and Edison Electric Institute).

Our key points are:

1. The process used to prepare the Draft is neither an appropriate nor a reliable way to assess public health risks;
2. The Draft is neither consistent with the available science nor in agreement with other international reports prepared by independent experts;
3. The authors do not have the expertise in all of the relevant scientific disciplines to fully evaluate the EMF literature, such as expertise in laboratory experiments, whole animal bioassay, and biophysics;
4. The risk communication messages have not been tested and are likely to be confusing and misleading, particularly to the general public;

We provide an elaboration of these points in Appendix A. With respect to the 'policy evaluation' report, the CDHS policy options evaluation uses risk evaluation outputs that consistently overestimate risks from EMF exposures. In addition, 'EMF Mitigation' costs are consistently underestimated. The result is an erroneous policy options evaluation that grossly overestimates the value of EMF "mitigation" measures, particularly with regard to 'property value impacts' and undergrounding of electrical facilities. Decision-making based on these faulty assumptions will adversely impact the siting of new electrical facilities, construction schedules, ratepayer costs, and electrical system reliability.

Unless the deficiencies in the report are effectively addressed, our common goal of developing appropriate and sound public policy on EMF in California, factoring in both public health and public utility considerations, will be made more difficult and will be substantially delayed. For CDHS, this may mean a loss of confidence in your agency's ability to assess public health risks, an inappropriate consideration of EMF risks relative to other health risks, as well as the larger concern of establishing a precedent of using a flawed risk assessment process for the evaluation of other health risks. For the CPUC, flawed decision-making may adversely impact the operation and costs of the electricity supply system by, for example, making it more difficult to site new electrical facilities that will be needed to connect new generation facilities to the grid. For utility customers, flawed decision-making will increase the costs of electricity without adding corresponding value to consumers.

We are recommending that CDHS:

1. Broaden the authorship to include perspectives from independent scientists who have working knowledge of relevant epidemiology, laboratory experiments, whole animal bioassays, and biophysics;

2. Carefully review the comments from independent scientific experts, and use this information to revise the report so that it reflects the existing EMF literature;

3. Increase the quality of 'Peer Review' independent of CDHS (and with more involvement of the CDHS EMF Science Advisory Panel).

The California electric utilities have been leaders in establishing an effective, proactive California EMF program. We have supported EMF research since 1979; performed health studies of our workforce; helped sponsor studies of childhood cancer in our service area; worked with the California Department of Education in the siting of new schools; actively communicated with our customers; and since 1992, adopted a policy of choosing options to lower magnetic fields from new electric utility facilities. CDHS has been an important partner over these past fourteen years. We believe that, moving forward; we can and should continue to work together to respond to the scientific uncertainty associated with the EMF issue by establishing sound and responsible EMF policies. To do this, California decision makers need the CDHS EMF reports to be technically correct, to be consistent with the available scientific information, and to communicate the key issues fairly in a way that is useful to the public and California decision makers.

Specific responses to the specific questions raised in the conclusion of the Draft are provided in Attachment B. Please contact us if you would like additional information to support our recommendations.

Sincerely,

John Dawsey
San Diego Gas & Electric

Randy Erickson
Modesto Irrigation District

Michael Herz
Pacific Gas & Electric

Enrique Martinez
Los Angeles Department of Water and Power

Kent Jaffa
PacifiCorp

Kuldip Sandhu
Sierra Pacific Power

Jon Sirugo
Southern California Edison

cc: Mr. Paul Clanon, CPUC
Ms. Judith Ikle, CPUC

Appendix A. Elaboration On Summary Points

1. **The process used to prepare the Draft is neither an appropriate nor a reliable way to assess public health risks**. While innovative, the 'Risk Evaluation Guidelines' used by CDHS do not represent established practice for public health risk assessment. You also used a procedure modeled on an 'International Agency for Research on Cancer (IARC) Monograph Review', but is substantially different from the formal IARC process. Importantly, however, the IARC procedures are not designed to provide a formal risk assessment. Second, the failure to use the same method as employed by IARC makes it inappropriate to suggest that an IARC-type assessment has been completed. Third, in order to allow objective assessment of the report's conclusions, the key differences between the CDHS process and the IARC process (which convenes an international panel representing a wide range of scientific disciplines) should be made explicit. Fourth, the Bayesian method that was originally intended requires a level of quantification that cannot be achieved for EMF, because key information is lacking (e.g., there is no reliable way of assigning 'exposure' or assessing 'dose').

2. **The Draft is neither consistent with the available science nor in agreement with other international reports prepared by independent experts**. The authors' main argument is that since exposure has not been proven to be safe, this increases the likelihood that EMF is a health hazard. This is tantamount to turning "ignorance into knowledge." Since the absence of causation is virtually "unprovable" for most environmental agents, use of this line of reasoning sets dangerous precedent for the evaluation for this and other public health issues addressed by the CDHS. Too much weight is given to the epidemiological results, and too little weight is given to the results from laboratory studies, whole animal bioassays, mechanistic studies and physical theory. Many of the conclusions reached by the authors are not scientifically supportable and are not in line with the conclusions of other scientific bodies. In fact, the conclusions in the Draft are at odds with those of all other risk assessments conducted by state, national and international agencies and major scientific organizations. This includes recent assessments by agencies with access to the same data (e.g., Virginia Dept of Health, National Radiological Protection Board (UK), the Health Council of the Netherlands, and the World Health Organization's International Agency for Research on Cancer).

   For example, CDHS used the criteria of the World Health Organization's International Agency for Research on Cancer (WHO/IARC) to classify EMF; they came to substantially different conclusions. With respect to childhood leukemia, the three DHS reviewers classified EMF risk as "possible," "probable," and "virtually certain". Since the time DHS completed its evaluation, the actual 20-member IARC panel reviewed the same EMF data and unanimously classified EMF risk for leukemia "possible," not probable or likely (IARC Press Release for Monograph 80, July 2001). Further, IARC found no consistent evidence with regard to all other childhood and adult cancers.

3. **The authors do not have expertise in all of the relevant scientific disciplines (expertise in laboratory experiments, whole animal bioassay, and biophysics needs to be added).** The CDHS committee that prepared the draft report is not representative of the wider scientific community with expertise in this area. The three authors are all from the same discipline (i.e., epidemiology), work in the same division, and two of the authors report to the third. In addition, for the miscarriage risk assessment, the suggestion for a problem comes only from recently published reports funded and written by these same individuals. While we are not impugning the qualifications of these scientists, a proper review of the extensive literature needs a broad range of expertise and institutional affiliations.

4. **The risk communication messages have not been tested and are likely to be confusing and misleading**. As currently drafted, this will pose particular problems with the general public. This confusion may inappropriately shape individual views of EMF risk and result in inappropriate public policy decisions. It should be recognized that there are adverse public health consequences from over-estimation of risk as well as from under-estimation of risk. The point is that CDHS's risk communication methods are not policy-neutral, and are likely to result in a substantial over-estimation of public health impacts. This, by itself, can adversely impact public health priorities.

Appendix B. Response to Specific Questions Raised by CDHS

CDHS specifically requested answers on the following questions:

1. We have taken the position that we are not greatly influenced by arguments based on physics and simplified biological models that suggested that residential and occupational levels of EMFs can't possibly produce bio effects. We say that theories should be used to predict results that are falsifiable and should not be used to discount evidence. Thus, our prior degree of confidence is not vanishing small. Do you agree? Please comment.

   Our Response:

   > We disagree with your use of biophysical evidence. First you misconstrue the argument, saying that since 'biophysics does not prove that EMF is safe, then this stream of evidence is not valuable'. You also discount biophysical theory by over-looking the substantial base of direct, reproducible experimental 'observation' that was used to construct these theories. The point isn't that your prior degrees of confidence are too low; it is that the three reviewers give too much weight to a highly selected set of 'new epidemiological information'. You fail to recognize the added importance of biophysical plausibility when the epidemiology conclusions are based on small numbers and weak effects, and no specific magnetic field parameter has been identified. In sum, you have consistently underestimated the value and relevance of the established biophysical theory in your evaluation of the epidemiological data and the whole animal bioassays to your risk assessment.

2. Each of the three core reviewers has laid out their initial (prior) degree of confidence that residential or occupational EMFs could produce relative risks of various sizes. These estimates are constrained by what we know about animal bioassays for cancer and by the lack of dramatic change in disease rates after the introduction of electricity and as the use of electricity increased. Reasons are given for these judgments. Do they seem reasonable? How much higher or lower would your priori degree of confidence be for any environmental/occupational agent? For EMFs? Why?

   Our Response:

   > Your method of risk assessment is neither scientifically sound nor defensible. You have wrapped yourself in Bayesian methods, without actually performing Bayesian analysis. First, the CDHS draft report did not follow the procedures outlined in the CDHS EMF Risk Evaluation Guidelines. Second, the risk assessment methods used are not considered standard practice for evaluation of potential public health risks. Third, the methods used are not useful for performing scientific risk assessments. CDHS should use established risk assessment methods. CDHS should also increase the number of authors by including scientists with expertise in the disciplines that are relevant to the available scientific information. This also will help to make the assessments more representative of the wider scientific community and to improve the relative weighting of data from the various scientific disciplines (e.g., laboratory experiments, whole animal bioassay, epidemiology, and biophysics).

3. We were not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects nor were we convinced of a chain of events that led to pathology. Yet we did not let this pull our degree of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment.

   Our Response:

   > No, we do not agree with your analysis. You have missed the key point. The important aspect of the available scientific literature is not that there is no established biophysical mechanism for the health risks suggested in the epidemiological literature (even though scientists have looked for such a mechanism for many years). Rather, it is that there are very well accepted biophysical mechanisms for the interaction of ELF/EMF and human cells. This is supported by the vast experimental literature and

the results of numerous, and relevant, whole animal bioassays. The epidemiological data are less plausible given this available knowledge. In addition, your confidence in the epidemiology is misplaced. While the epidemiological literature can be described as 'limited,' we do not believe that, as scientists, you can confidently assert that we can rule out bias, confounding, or chance as plausible explanations for the observed associations in the pooled analysis (for exposures above 4 mG).

4. We viewed the animal pathology literature as largely null, with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment.

Our Response:

You are alone in viewing the Loescher work as either relevant to cancer promotion or of value in addressing the question of potential health risks. The published reports from Loescher do not support any 'effect', the studies were not replicated, and there are results from other, well-designed and conducted studies that do not show any health effects. Your reliance on the 'Henhouse' studies is inappropriate. In 1997, a group of experts including two of the DHS reviewers unanimously concluded that the chick assay studies are equivocal and not a good assay for human risk assessment. The exposures for these studies are also not relevant to those found in community or occupational environments. You discount the lack of results from the majority (and best designed and conducted studies) of the animal bioassays by creating vague theories of disease causation. Even though these vague theories of causation were not addressed in the available literature, and you have no data to support them, you assume that the 'theory' supports the epidemiological literature.

5. Not all epidemiologists would agree with our position that relative risks between 1 and 2 should be taken seriously unless there is specified evidence for confounding or bias to explain it away. Do you agree? Please comment

Our Response:

We disagree. We know from experience that there is a poor predictive value of epidemiological results for low estimated Relative Risks (e.g., review the contents of *American Journal of Epidemiology* or *Epidemiology* over the last ten years for studies that report estimated RR at these levels and note how the results are described). Your view is especially flawed in the context that there are small numbers of high exposed subjects and there is a lack of biophysical, experimental and animal support. With regard to small numbers, the pooled analysis by Ahlbom *et. al.,* reports that only 0.8% of subjects had exposure above 0.4 μT. The large majority of these subjects come from the study by Linet *et. al.,* who have demonstrated that participation bias and confounding occur in this study. In addition, no specific exposure parameter has been identified. In such cases, it is inappropriate to over-interpret the epidemiology.

6. We said that a lack of specificity in the association of EMFs with subtypes of cancer and evidence for effects on various types of disease did not pull down our degree of confidence and might even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature. Do you agree? Please comment.

Our Response:

We disagree. It is implausible that EMF is a 'general health hazard.' First the scientific data do not support this (e.g., neither laboratory experiments nor whole animal bioassays find robust suggestions for adverse effects on intact cells or tissues). Second, if EMF were a 'general health hazard,' this would imply that the disease model would be more conspicuous, which would suggest the whole animal bioassay and the laboratory experiments would find more robust results. This should either be neutral to your weight of evidence or diminish your confidence. Over the last thirty years, a vast number of exposures and different

disease types have been evaluated. None of the earlier suggestions for an effect, including the '2 mG MF level' suggested by Wirtheimer and Leeper (1979), have held up to better studies. In contrast, Reviewer 1 uses this line of thinking to increase his belief that EMF is linked to health impacts. There is no evidence for a common biological model between the six diseases that Reviewer 1 concluded were likely to be caused by EMF exposure (i.e., childhood leukemia, adult leukemia, adult brain cancer, female breast cancer, spontaneous abortion and ALS). While Reviewer 1 concludes that he is virtually certain that EMF exposure is not a 'Universal Carcinogen', he does maintain that three fundamentally different cancer sites are linked to EMF exposure. A fair reading of the available scientific data does not support this.

7. Have we done an adequate job in presenting the arguments for and against causality or are we assigning weak arguments to the "con" or the "pro" position?

Our Response:

You have presented the arguments, but you fail to assign sufficient value to the 'con' arguments and give too much credit to the 'pro' arguments. The analysis also lacks scientific rigor and does not give sufficient weight to key aspects of the scientific literature.

8. Our Risk Evaluation Guidelines (REGs) define some "plain language phrases" to express our degrees of confidence. However, when we actually applied them we found they were not problem free:

a) Some of these phrases are not mutually exclusive. For example, Possible >50% overlaps "highly probable" and virtually certain." "Possible <51%" overlaps "Possible >50%". In this case, the overlap is slight, but important, since it is about the "balance of probability".

b) These phrases are grammatically awkward and they are not really "user friendly". How could we rephrase them, without violating the spirit of the REGs? Please write any suggestions next to each phrase:

Confidence range	Current Phrase	Suggested alternative
>98%	Virtually certain	
90-98%	Highly probable	
50-90%	Possible >50%	
10-50%	Possible <51%	
2-10%	Very improbable	
<2%	Virtually certain that it is not causal	

Our Response:

There is no scientific justification for these categories. These are not consistent with the text used to describe the assessments of any other independent expert panel. For example, based on the same epidemiological data, the National Institutes of Environmental Health Sciences (NIEHS) concluded that:

> The scientific evidence suggesting that ELF-EMF exposures pose any health risk is weak.

> The NIEHS concludes that ELF-EMF exposure cannot be recognized at this time as entirely safe because of weak evidence that exposure may pose a leukemia hazard.
>
> The National Toxicology Program routinely examines environmental exposures to determine the degree to which they constitute a human cancer risk and produces the 'Report on Carcinogens' listing agents that are 'known human carcinogens' or 'reasonably anticipated to be human carcinogens'. It is our opinion that based on evidence to date, ELF-EMF exposure would not be listed in the 'Report on Carcinogens' as an agent 'reasonably anticipated to be human carcinogens.'
>
> *NIEHS Director's EMF-RAPID Report to Congress, June 1999*

For an international perspective, the U.K. National Radiation Protection Broad (NRPB) has a standing committee, chaired by an eminent epidemiologist, Sir Richard Doll. This committee has concluded:

> In the absence of any unambiguous experimental evidence to suggest that exposure to these electromagnetic fields was likely to be carcinogenic, the Advisory Group concluded that the findings of the epidemiological studies that had been reviewed could be regarded only as sufficient to justify formulating hypotheses for testing by further investigation. They provided no firm evidence of a carcinogenic hazard to either children or adults from exposure to normal levels of power frequency electromagnetic fields.
>
> *National Radiation Protection Board, United Kingdom, March 2001*

Regarding question 8 (above), CDHS should use statements that are similar to those of NIEHS or the U.K. NRPB. The scientific validity and reliability of expressing your views of risk based on 'categories of percentage likelihood' have not been established. If CDHS must use this approach, the full confidence range from the lowest of the low to the highest of the high should be indicated. For example, rather than stating an exact "xxxx", use a range, like, "It is between '20% and 90% likely' that EMF's at home or at work could cause a very small increased lifetime risk of childhood leukemia, adult brain cancer, and Amyotrophic lateral sclerosis (ALS, Lou Gehrig's Disease)."

We would also ask that CDHS provide appropriate framing for the chosen text. For example, a statement such as the following would be appropriate:

> The percent confidence intervals are not formal scientific assessments, but are estimates generated for the purpose of using computer modeling to evaluate policy options. Because their validity and reliability have not been established, they should not be interpreted as pertaining to the actual likelihood of disease for either population groups or individuals within the exposed populations.

August 12, 2001

Dr. Raymond Neutra
Chief
Division of Environmental and Occupational Disease Control
Department of Health Services
California EMF Program
1515 Clay Street, Suite 1701
Oakland, CA 94612

Dear Dr. Neutra,

Attached you will find my comments on draft 3 of the California EMF Program – Risk Evaluation. I have reiterated points that I made at the meeting earlier this month and provide my thoughts on a few other issues. It thought the meeting was productive and it certainly highlighted the diverse opinion of the panel and it is should be very clear to the public that the panel was not "stacked" one way or the other. Please let me know if your have any questions or need clarification on any points.

Sincerely,

Michael S. Denison, Ph.D.
Professor of Environmental Toxicology
University of California, Davis
4138 Meyer Hall
Davis, CA 95616

My principal concerns with the document were related to the lack of details and references in the mechanism section and the issue of the level of confidence that derives from the mechanistic studies. In section 5.0 In Vitro Mechanistic Evidence, I was concerned by the total lack of any referenced papers and/or results (although I note that none were provided for the biophysical section as well). I had to go through the entire list of references at the end of the documents to find the 2 or 3 papers that were actually related to this section. I also noted that a brief review of the literature using online databases provide a significant amount of new papers (i.e. published after the NIEHS study) which should be examined and considered since the California EMF review is using the most recent results. I strongly feel that some description of the mechanistic results that you are using to support your degree of confidence in an EMF effect needs to be provided. As it stands, a reading of this section really provides no insight into the "mechanistic studies" that have been done and their consistencies and/or inconsistencies. Additionally, if the chicken results are going to be discussed in any meaningful way then a brief discussion of the variation in response between different strains (i.e. the different strains of chickens as well as differences between white leghorn chickens bred for food and those for eggs) needs to be included. Although the effects of EMF at ambient levels is clearly the issue, if studies do provide consistent biological effects using higher EMF levels, then this should be discussed. The fact that consistent effects are observed even with these high levels would provide good support that EMF-dependent biological effects can occur. However, the absence of consistent results even with high EMFs would dramatically reduce my confidence that EMF would have any effect, especially at low levels. Given that the conclusion of the in vitro results section is that the results are inconclusive, the comments on page 206 (lines 22 and 23) that states that the replicated animal and in vitro studies at low exposure provide additional confidence is not justified.

There are also other mechanistic issues that I think complicate clear consideration of EMFs as causative agents in adverse health effects. These issues stem from the apparent lack of dose response effects in both In Vitro and animal studies and the lack of consistent effects between studies. I am concerned by the fact that positive results presented in one publication would greatly increase the confidence levels of the reviewers that EMF could produce adverse effects, yet negative results would not affect their confidence levels much. In my mind the reviewers would need to assume that the negative studies were wrong or incorrectly done, while the positive studies were correct and appropriately run. The fact that some very large and well-designed studies have yielded little positive EMF effects dramatically reduces my confidence level. I don't think that we can simply discard these negative studies and assume that the ones reporting positive effects are the only correct ones. Accordingly, I am not convinced by the results presented and referenced in the document that EMFs are a causative agent in adverse health effects. Given my above comments, I think that I would also agree with the comment by Jim Cleaver that there needs to be some clear justification as to why the minimal confidence limit wouldn't go to zero.

I also felt that that the analogy of EMFs to chemical agents is not appropriate. There is clear evidence that effects of chemicals on cells is dependent upon different biological targets (i.e. receptors) which are differentially expressed between cell and tissue types. EMF on the other hand is a biophysical agent that would be expected to produce some common biological effect in a cell, although the ultimately observed effect may vary and could be dependent upon the cell type. Comparisons to another biophysical agent like ionizing radiation may be a more appropriate analogy.

Page 8 (48-60) versus 16 (line 54-58). This is a concern I had previously that was not altered. I still agree that the definition of the degree of confidence is a significant point and the description on page 8, lines 48-51 makes this point very clearer. However, given this, I still am confused as to why you use the degree of confidence to then estimate the number of persons (i.e. "expected number") who would get EMF related disease (see page 16, lines 54-58). What this should say is that we are 30% confident that some of the cancers observed in these individuals are due to EMFs. This sentence should read very similar to what you have stated on page 8, lines 58-60.

Page 8, line 66-68. Shouldn't this say "fact" or "agent" X instead of "disease" X?

The references to the EMF-miscarrage paper of Lee et al., 2001 and Li et al., 2001 that are in press, should be listed in the document as 2001 and not 2000. The dates were changed in the most recent reference list but not the document itself. I also noticed that many references were incomplete and this needs to be corrected.

Jack Collins: I believe that the current proposed study is redundant of previous studies,unbalanced,and wasteful of tax payers monies. Tell me, who gets paid in this situation?
You may contact me at websbd1@hotmail.com

Dear Mr Collins,

This report and 2 mG recommended threshold are of great importance to the future health of Californians.

Not addressed in the report, however, were potential effects of EMF to citizens who were sick or suffering of chronic illness. In reviewing the NIEHS website for effects of sub 10 mG magnetic fields, their published research reports (scientists Blumenthal, Portier, McMillian, Lai) concluded with such results as lowered melatonin levels, decreases in cholinergic activity, and alteration of neuro-immune function. Research reports from one of NIEHS's labs, IITRI Life Sciences, concluded that sub 10 mG magnetic fields reduce NK cell activity (House and McCormick).

Given the results of these studies:
1) Would it not be apparent that persons suffering from lupus, AIDS, chronic fatigue syndrome, etc. be especially susceptible and especially affected by low magnetic fields under 10 mG and even under 2 mG?
2) Can and should a medical doctor recommend lowered EMF environments (e.g. below 2 mG) to patients who are suffering chronic illness?
3) Would it not be apparent that persons suffering from mental illness, especially depression, may be especially susceptible to low-level magnetic fields?

There is a small but substantial population of people in California who are suffering from these types of illness. These are groups of persons who need to be represented by the PUC EMF study. More importantly, they may benefit their health by changing their environment, that is if EMF were to have a more negative effect on them, in either the short or long term, than on a healthy person.

I look forward to your responses.

Sincerely,

Scott Dykes
scottdykes@yahoo.com

EPRI Comments on the California Department of Health Services Preliminary EMF Risk Evaluation

The California Department of Health Services (CDHS) has released its public-comment draft evaluation[1] of the possible health risks from exposure to electric and magnetic fields (EMF) from power lines, indoor wiring, electrical occupations, and appliances. The draft report is a product of the California EMF Program, a research and policy analysis effort mandated in 1993 by the California Public Utilities Commission. Along with the risk evaluation, CDHS has released a summary of policy options[2] to deal with possible risks from EMF exposure from power grid sources and in schools. The policy options summary will be the subject of another set of *EPRI Comments*. CDHS welcomes public comments on both documents before noon Pacific Time, September 10, 2001. EPRI intends to submit more detailed comments to CDHS on both documents.

Risk Evaluation Procedure

Three CDHS scientists from the California EMF Program were assigned to review the studies on health problems possibly related to EMF exposure. They considered all studies available for review by June 2000. For studies published before 1998, the National Institute of Environmental Health Sciences (NIEHS) Working Group Report (1998)[3] was taken as the starting point. The evaluation was based on biophysical theory; research on mechanisms by which EMF might produce physiological effects; experiments in laboratory animals; and epidemiologic studies.

The CDHS reviewers performed two separate evaluations. First, they used risk evaluation guidelines previously developed by the EMF Program to arrive at a "degree of confidence" that exposure to EMF could causally contribute to 13 different health outcomes previously linked to some surrogates for EMF exposure in epidemiologic studies. Second, they classified the evidence for all outcomes according to International Agency for Research on Cancer (IARC) criteria. These criteria classify substances or exposures according to weight of evidence of carcinogenicity as (1) carcinogenic to humans, (2) probably carcinogenic, (3) possibly carcinogenic, (4) not classifiable as to carcinogenicity, and (5) probably not carcinogenic. CDHS used these criteria for non-cancer outcomes as well as cancer outcomes. (For a more detailed explanation of IARC criteria and a summary of the 2001 IARC evaluation of the evidence on the carcinogenicity of EMF, see *EPRI Comments*, June 2001[4]).

The completed evaluation was submitted to a panel of 10 CDHS scientists and then to a Science Advisory Panel of outside scientists for further review. The current draft document is open to public comment, to be considered for incorporation into a final version.

CDHS Conclusions

Based primarily on epidemiologic evidence, the reviewers reached the following conclusions in their degree-of-confidence evaluation:

- It is "more than 50% possible" that residential or occupational EMF exposure could cause a very small increased lifetime risk of childhood leukemia, adult brain cancer, and amytrophic lateral sclerosis (ALS, or Lou Gehrig's disease). However, there is a chance that EMF have no effect at all.
- It is "more than 50% possible" that residential or occupational EMF exposure could cause a 5–10% added risk of miscarriage; there is also a chance of no effect.

- It is "10–50% possible" that exposure to EMF could cause a small increased lifetime risk of male breast cancer, childhood brain cancer, suicide, Alzheimer's disease, and acute myocardial infarction (heart attack). It is "at least 10–50% possible" that EMF exposure could cause a small increased lifetime risk of adult leukemia and female breast cancer. Again, there may be no effect.

- It is "2–10% possible" (very unlikely) that EMF are universally carcinogenic or that EMF cause reproductive and developmental problems such as birth defects, low birth weight, or neonatal deaths.

The reviewers' classification of EMF according to IARC criteria is as follows:

- Between possibly carcinogenic and carcinogenic in humans for childhood and adult leukemia. (This classification is based on three different conclusions given by the three reviewers. Reviewer 1 classified EMF as carcinogenic for both childhood and adult leukemia; Reviewer 2, as possibly carcinogenic for childhood and adult leukemia; and Reviewer 3, as probably carcinogenic for childhood leukemia and possibly carcinogenic for adult leukemia.)

- Possibly a cause of adult brain cancer, ALS, and miscarriage.

- Evidence is inadequate to determine whether exposure to EMF increases the risk of male or female breast cancer, childhood brain cancer, suicide, Alzheimer's disease, acute myocardial infarction, cancer in general, birth defects, low birth weight, neonatal deaths, depression, and electrical sensitivity.

The reviewers explain that evaluation of EMF on the basis of the IARC system was sometimes inconsistent with the degree-of-confidence evaluation. Unlike IARC, CDHS provided explicit pro-and-con justification for its classifications and also considered yet-to-be-published evidence excluded by the IARC system.

The proportion of childhood leukemia cases possibly attributable to EMF has been estimated at about 4% per year, or, in California, about 4 deaths per year. The reviewers estimate that if the same 4% were applied to the other 11 conditions evaluated as not "very unlikely" to be caused by EMF, the numbers of attributable cases would be in the hundreds or thousands and comparable to numbers of cases that have motivated other environmental regulation. For environmental exposures, many regulatory processes are triggered by a benchmark figure for theoretical added lifetime risk of above 1/100,000. (For occupational exposures, the figure is 1/1000.) The reviewers concluded that since even the lowest detectable risks in epidemiologic studies involve lifetime risks greater than 1/100,000 for even rarely occurring diseases, these risks, if real, could be of regulatory concern.

With regard to miscarriages, the reviewers reported that two new, yet-to-be-published epidemiologic studies[5,6] suggest that short, very high EMF exposures probably originating from appliances and indoor wiring (rather than from living near power lines) may be responsible for a substantial proportion of miscarriages (40%) in California. Because miscarriages are common, the theoretical added risk from EMF exposure would be of regulatory concern. The reviewers stressed, however, that the majority of exposed women in the studies did not miscarry. Finally, the CDHS evaluators noted that scientific research progresses slowly and recommended a long-term commitment to research funding to resolve the EMF question. According to a cost-benefit analysis, the difference in the cost of various policy options for EMF mitigation (expensive, inexpensive, or no mitigation), the choice of which is driven by scientific uncertainty, is so large that more research funding can easily be justified to reduce this uncertainty. The evaluators recommended further research in several areas of EMF science. Of high priority are residential and occupational exposure assessment studies to determine if "aspects of the EMF mixture . . . such as micro shocks, stray ground current, and charged air pollutants," might explain any apparent effects. Other research priorities include more common health outcomes for which possible EMF effects could have a substantial public health impact: myocardial infarction, spontaneous abortion, and suicide.

EPRI Comments

The CDHS risk evaluation differs in several ways from other recent evaluations by NIEHS, the National Radiological Protection Board (NRPB)[7] in the UK, and IARC[8]. The use of the IARC classification scheme by the NIEHS Working Group and CDHS simplifies comparisons between evaluations by these three groups. Of note are the following differences:

- IARC classified EMF as possibly carcinogenic, mainly based on childhood leukemia studies, and the NIEHS Working Group Report classified EMF as possibly carcinogenic for childhood leukemia and chronic lymphocytic leukemia in occupationally exposed adults. CDHS classified EMF as between possibly carcinogenic and carcinogenic for childhood and adult leukemia; this classification reflects the range of conclusions given by the three reviewers. NRPB, which did not use IARC criteria, concluded that there is a possibility that EMF may increase the risk for childhood leukemia.

- CDHS accorded a possibly causative classification to EMF for adult brain cancer, ALS, and miscarriage. The NIEHS Working Group found the evidence for these conditions inadequate for classification, and NRPB reported that the evidence does not point to an EMF association. IARC evaluated only cancer and did not consider other diseases, which will be reviewed by the World Health Organization (WHO) in an upcoming evaluation.

- Unlike any of the other groups, CDHS used a quantitative evaluation method based on degree of confidence of an effect rather than a qualitative classification system alone. This is the first time this type of approach has been applied to evaluations of possible EMF health effects.

- CDHS included new evidence not used in previous evaluations, including results from two yet-to-be-published studies[5,6] on miscarriage. These studies weighed heavily in the reviewers' classification of EMF as a possible cause of miscarriage. All previous evaluations found no increased risk for miscarriage.

CDHS, like NRPB and NIEHS, stressed the need for further research. IARC did not make research recommendations, although its classifications will help guide research priorities. While the 2001 NRPB report recommended additional research in biophysics, cellular and animal studies, and residential and occupational epidemiologic studies, CDHS emphasized the importance of research on exposure assessment and on more common health outcomes (myocardial infarction, spontaneous abortion, and suicide) that could have a large public health impact.

EPRI is conducting research—with diminished funding, as noted in the CDHS report—in several areas recommended by CDHS and other groups. Current EPRI research includes cellular studies, contact current studies, childhood leukemia work, and an occupational epidemiologic study of cardiovascular outcomes.

For further technical information, please contact:

- Rob Kavet at 650-855-1061 (rkavet@epri.com)

Press contact:

- Christine Hopf-Lovette at 650-855-2733 (chopf@epri.com)

References

1. California EMF Program. An evaluation of the possible risks from electric and magnetic fields (EMFs) from power lines, internal wiring, electrical occupations and appliances. Draft 3 for public comment. California Department of Health Services (CDHS). Online. Internet. April 2001. Available at http://www.dhs.ca.gov/ehib/emf/RiskEvaluation/riskeval.html.

2. California EMF Program. Policy options in the face of possible risk from power frequency electric and magnetic fields (EMF). Draft for public comment. California Department of Health Services (CDHS). Online. Internet. April 2001. Available at http://www.dhs.ca.gov/ehib/emf/RiskEvaluation/riskeval.html.

3. National Institute of Environmental Health Sciences (NIEHS). Assessment of health effects from exposure to power-line frequency electric and magnetic fields. NIEHS Working Group Report. Research Triangle Park (NC): National Institute of Environmental Health Sciences; 1998. NIH Pub. No. 98-3981.

4. EPRI. EPRI comments on the IARC Working Group's evaluation of the evidence of the carcinogenicity of EMF. Online. Internet. June 2001. Available to EPRI EMF Health Assessment customers at http://www.epri.com.

5. Lee GN, Hristova L, Yost M, Hiatt R. A nested case-control study of residential and personal magnetic field measures and miscarriages. *Epidemiology* 2001, submitted.

6. Li D-K, Odouli R, Wi S, Janevic T, Golditch I, Bracken T, Senior R, Rankin R, Iriye R. A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of spontaneous abortion. *Epidemiology* 2001, in press.

7. National Radiological Protection Board (NRPB). ELF electromagnetic fields and the risk of cancer. Report of an advisory group on non-ionising radiation. Oxon: Doc. NRPB 2001; 12(1):1–179.

8. International Agency for Research on Cancer (IARC). Static and extremely low frequency magnetic fields. Vol. 80. Online. Internet. 18-26 June 2001. Available at http://monographs.iarc.fr.

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August 25, 2001

Dr. Raymond Neutra
California EMF Program
1515 Clay Street, Suite 1700
Oakland, California 94612

Attention: Jack Collins

Regarding: The Risk Evaluation

Dear Dr. Neutra,

On Wednesday, August 22 at 1 p.m. I participated in a 2-hour telephone conference with you and Dr. del Pizzo. Ms. Susan Molloy, Mr. Ken Ceder, and Mr. John Dawsey (of San Diego Gas and Electric) also participated. I represent the Cellular Phone Taskforce, a national organization whose membership includes hundreds of electrically sensitive people.

A substantial portion of the telephone conference concerned electrical sensitivity. This letter, with enclosures, is written both to respond to questions you put to me during the conference, and to put on paper my recollection of what was said on Wednesday.

First, you asked me what additional studies I think should be included in the literature review in Appendix Four. I am enclosing a number of such studies. These are some comments about them:

There are really two facets to examine: (a) How does electricity affect health? (b) Are some people more sensitive than others? When looked at this way, the questions are less controversial, and the answers are easier. The answer to (b) is obviously "yes'. In medical school, when I learned about any disease,, they always taught me what the risk factors were and what segments of the population were more susceptible. Electrical illness can't be any different. There are always predisposing genetic, occupational and other factors which determine one's susceptibility to biological stressors.

Therefore I recommend the term "electrical sensitivity" (rather than "hypersensitivity") to indicate a whole range of sensitivity. "Hypersensitivityllseems to imply that there is a qualitative, rather than a quantitative, difference in human reaction to electricity. When such a qualitative difference is not found, many researchers have concluded that ES does not exist. But they have made an a priori, and wrong, assumption about what they were going to find: namely, that we ES are something other than human.

The Risk Evaluation asked questions (a) and (b) separately. Its failure to make a connection between the two impaired its ability to answer either one. Specifically, in Chapters 13 through 18, you looked at possible reproductive, neurological,, cognitive, cardiological, and emotional outcomes of exposure in the normal population. Reproductive, neurological,, cognitive,, cardiological and emotional outcomes in the ES population can shed light on this. Conversely, attention to objective neurological, cardiological, cognitive, emotional and

reproductive signs and symptoms can help experimenters define ES objectively and choose patient and control populations in a more rational manner when designing double blind studies.

That said, it becomes clearer where to look for better information about ES. First, one should look at highly exposed populations, especially occupationally exposed populations. Second, one should look at data on the effects of natural electricity, i.e. at the literature on "weather sensitivity". Third, one should look at studies of ES populations which were designed to collect physiological data,, not just percentage of right guesses as to when the electric field is on or off. Fourth, because electric currents induced to flow in the human body should have similar effects regardless of what induces them to flow, therefore data on RF is just as relevant as data on power frequencies to the nature of ES. However, I am not including any RF studies in this packet because you asked me not to. Enclosed are:

SOVIET STUDIES

1. F.A. Kolodub and O.N. Chernysheva, "Special Features of Carbohydrate-energy and Nitrogen Metabolism in the Rat Brain Under the Influence of Magnetic Fields of Commercial Frequency", Ukrainskiv Biokhimicheskiy Zhurnal No. 3,, 19800 pp. 299-303. JPRS 77393.

2. Jiri Jerabek et al., "Biological Effects of Magnetic Fields", Pracovni Lekarstvi 31(3):98-106, 1979. JPRS 76497.

3. "Effect of a Constant Magnetic Field on Blood Clotting Indicators". Izvestiva Akademii Nauk Latviyskoy SSR No. 1. 19831, pp. 84-91. JPRS 84527.

4. .M. Serdyuk and L.G. Andriyenko, 'Effect of Electro-magnetic Energy on Generative Function of Animals", Doklady Akademii Nauk Ukrainskoy SSR. Seriya B. Geologicheskive, Khimicheskiv i Biologicheskiye Nauki No. 6. 1983, pp. 76-79. JPRS 84527.

5. T.P. Asanova and A.I. Rakov, "The State of Health of Persons Working in the Electric Field of outdoor 400 and 500 kV Switchyards", Study in the USSR of Medical Effects of Electric Fields on Electric Power Systems, Special Publication Number 10 of the Power Engineering Society, IEEE 1975.

6. T.E. Sazonova, A Physiological Assessment of the Work to Conditions in 400-500 kV Open Switching Yards", Ibid.

The above are all from the former Soviet Union. #1 is an animal study revealing effects on metabolism and respiration. #2 establishes symptoms observed in workers: headaches, vertigo, buzzing in the ears, chest pains, irritability, bradycardia, hypotension, perspiration, abdominal pain, digestive problems, tremors, altered reflexes,, muscle and joint pain, weakness, inability to concentrate,, memory problems--in short, all the symptoms we complain about in E.S.--together with objective signs. Health effects were only found in 18-41% of the workers, depending on the intensity of the occupational exposure, showing a marked difference in individual sensitivities.

#3 verifies what the ES know: many of us have a tendency to spontaneous nosebleeds and/or internal bleeding, and some of us die of cerebral hemorrhage. #4 is an animal study showing effects on reproduction. #5 and #6 are more studies of workers,, again finding objective disease as well as the usual symptoms, i.e. headaches, fatigue, irritability, sleeplessness, palpitations, poor appetite, etc.

WEATHER SENSITIVITY

7. Felix Gad Sulman, M.D**.,** D.V.M., The Effect of Air Ionization, Electric Fields, Atmospherics and Other Electric Phenomena on Man and Animal, Charles C. Thomas, Springfield, IL 1980, pp. 11-12,127-1310 144-153t 192-196, 277-289.

This is a large book, so I am sending selected pages. Dr. Sulman says "We do great injustice to the electrosensitive patients, who rightly complain of their serotonin sufferings, when we treat them as psychiatric patients' (p. 12). 30% of any population are said to be weather-sensitive.

8. G. Ruhenstroth-Bauer et al., Epilepsy and Weather: a Significant Correlation between the Onset of Epileptic Seizures and Specific Atmospherics - a Pilot Study", Int. J. Biometeor. 28(4):333-340, 1984.

9. J. Juutilainen et al. , "Epilepsy and electromagnetic fields", Int. J. Biometeorol. 32:17-20, 1988.

10. Abstracts of 5 articles by M.A. Persinger on geomagnetism and epilepsy.

OTHER

11. Roy Fox and Roger Smith,, "Electromagnetic Sensitivity Triggers Environmentally Induced Dysfunction", in Proceedings of the 2nd Copenhagen Conference on Electromagnetic Hypersensitivity, May 1995, pp. 17-19. Dr. Fox notes that 6 of 20 ES patients were also extremely weather sensitive.

12. Indira Nair et al.,, Biological Effects of Power Frequency Electric and Magnetic Fields'. Office of Technology Assessment, Oct. 1990, pp. 50-51, Sec. 4.6 "Experiments with Human Subjects".

These authors cite studies by Deno and Zaffanella 1982-

Graham 1987; and Graham 1988, all of which found marked variation in individual sensitivity to electric fields, including the ability to perceive the fields**,** and effects on alertness, reaction time,, and heartbeat.

13. E. Stanton Maxey, M.D., "Critical Aspects of Human v. Terrestrial Electromagnetic Symbiosis",, in Biological Effects of Electromagnetic Waves,, Selected Papers of the USNC/URSI Annual Meeting, Boulder, Colorado, Oct. 20-23, 1975, Vol. 1, pp. 331-340.

This author found a phenomenon similar to photic driving, from weak ELF fields, in 2 of 13 subjects. The other 11 subjects showed no such response. He concluded that weak ELF fields can "control the brain's electrical rhythms in sensitive human subjects".

14. Olle Johansson and Peng-Yue Liu,'"Electrosensitivity, 'Electrosupersensitivityl and 'Screen Dermatitisl: Preliminary Observations from On-goingStudies in the Human Skin", pp. 52-57 of Proceedings of COST 244 Meeting on Electromagnetic Hypersensitivity, Sept. 26-27, 1994, Graz, Austria.

One of Dr. Johansson's papers was reviewed in your Appendix 4, pp. 94-95. This puts it in context of an ongoing program of research with many consistent findings.

15. Marek Szuba and Stanislaw Szmigielski, "Change in Reaction Time to Auditory and Visual Signals Differentiates Individual Response to Short-Term Exposure to ELF Electric **Fields** and Direct Current Stimulation", Id., pp. 94-104.

4 of 71 subjects reacted much more strongly to electric fields, and 2 of them also reacted to weaker fields than everyone else. The authors suggested using this as an objective screen for electrosensitivity.

16. Tong Wang, Leslie Hawkins, and William Rea, "Effects of ELF Magnetic Fields on Persons with Chemical Sensitivities", Id., pp. 123-132.

I am including this because on p. 105 of Appendix 4 Dr. Lavallois concluded that Dr. Reals group 'tried to reproduce [their] results with an improved design, but without success. (Wang et al., 1994,, reported by the Eur. Commission, 19971, and Leitgeb, 1998)". I wanted you to actually see the Wang study instead of relying on secondary sources to tell you what it supposedly says. By no stretch of the imagination is it a reproduction of Reals 1991 study. As I told you on the phone, I am myself critical of Reals study, but it is a totally different animal than the so-called reproduction by Wang et al. First, in the Rea study, the final patient group were selected experimentally by some objective means, so that 75% of the original volunteers were eliminated. The Wang "patient" population was a 100% self-selected group. Second, the Wang subjects were subjected to a magnetic field of 300 mG. approximately 100 times as large as the Rea subjects. As a person with ES I react to magnetic fields of more than 0.2 mG. If you blasted me with 300 mG for over an hour, with rest periods of only five minutes, like Wang did, I would have such a flow of adrenaline through my body that my nervous system would become non-reactive. The after-effects, though, would be terrible. Wang should have pre-selected his subjects, and also kept them overnight for observation.

17. Theodor Abelin, M.D.,, "Establishing Magnetic Fields as Health Risks to Humans and Animalsee , medicine meets Millennium, World Congress on Medicine and Health, Aug. 2. 2000.

Dr. Abelin took the same data base as Dr. Levallois, and came to totally different conclusions. Specifically, he found the literature review by Bergqvist in 1997 most useful to validating this problem, even though Bergqvist himself put a different spin on it. My conclusion agrees with Dr. Abelin's. See:

18. Paragraphs M and N of my affidavit to the High Court of Ireland in a case in which I was an expert witness in 1998. In paragraph M I pointed out the positive findings in the Anderson study, which the authors of the study missed (the study is discussed on page 104 of Dr. Levallois's Appendix 4). In paragraph N I pointed out that even though the European Commission's report in 1997 (written by the same Bergqvist) played down the problem, its own database,, contained in its Appendix 3,, overwhelmingly supports the reality of this problem.

In the process of commenting about the enclosed studies, I have already covered some of the points I made during our telephone conference and answered a few of your questions. In particular,, we discussed my opinion of existing double blind studies of ES, and I said the patients and controls should be chosen by objective means, not self-selection, which is unreliable and never used in studying any other disease. I said, further, that people are not machines, and that if you want to find out what an electric field does to them you should observe them at least overnight and record physiological data. You don't put them in a room for a few minutes and try and fool them. You can do that if you want to, but you won't get anything useful out of the experience. Also, better control of electrical exposure is critical. Too many experiments (including Dr. Reals) have been done with computers and fluorescent lights in the experiment room,, and with patient-monitoring equipment that is computerized and that itself affects people with E.S., confounding the results.

You also asked me about contact currents. This is similar to what they are still trying to do to the dairy farmers. The utility companies would like to pretend that if they fix the contact currents, the problem will go away. It won't. As I said to you on the phone, you can send a current through me by conduction, or by inductive or capacitative coupling, or by radiation, but it's the same current no matter how it gets into my body, and the biological consequences have to be the same.

However,, if you look at all of the existing evidence, there is plenty on which to draw a positive conclusion. Some of what is missing from Appendix 4 I have supplied in the enclosed packet. And some of what is wrong with Appendix 4 has to do with reliance on secondary, rather than primary sources, or on a double standard of evaluation for positive and negative studies. As I have shown in my evaluation of the Anderson study, it is as necessary to scrutinize carefully a negative finding as it is to scrutinize a positive one. And I might add, you should carefully inquire into conflicts of interest on the part of the authors. For example, Ulf Bergqvist,

whose name appears so frequently above literature reviews that are negative or "inconclusive". receives a substantial part of his income as scientific consultant to Swedish Telecom.

It also bugs me to see so much research discounted because the symptoms are "non-specific". "Non-specific" is a code word that has no scientific meaning and reveals only the prejudice of its user against the whole subject. I have looked into the history of this. The constellation of symptoms we are talking about was first described in 1869 by a Dr. George Beard, and has only come to be called "non-specific' because the medical community could not agree on a cause. If everyone could agree that the cause is electrical, then it wouldn't be "non-specific" any more, would it? There is a fiction going on here that it is somehow relevant that each particular symptom, e.g. headaches, is not specific but occurs in many diseases. Well, yes, for every symptom there is a differential diagnosis, no one symptom is specific to any disease. It's the constellation that usually clues you in. If somebody comes to me with pressure behind the eyes, pain in the testicles and the soles of the feet, and nosebleeds,, I have a high suspicion of electrical illness, probably radio wave sickness. Probably they are also not sleeping well, and having trouble with their memory. They may be dehydrated. Electrical sensitivity is a disease like any other.

I appreciated the time you gave Susan Molloy and me on Wednesday, and your attentiveness to what we had to say. I urge you to follow up by incorporating the enclosed documents into Appendix 4, and altering its conclusions accordingly.

Sincerely,

Arthur Firstenberg

Enclosures

Here is the text of a letter that is being mailed to you today.

****

UNIVERSITY of PENNSYLVANIA

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Professor of Bioengineering and Electrical Engineering
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September 7, 2001

Jack Collins
California EMF Program
1515 Clay Street, Suite 1700
Oakland, CA 94612
JCollins@dhs.ca.gov

Dear Mr. Collins:

I submit these comments about the draft report "An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances".

The approach of this report is very different from that normally taken by health agencies, which is to carefully weigh primary scientific data in search of evidence for a hazard. Indeed, very little of the voluminous scientific data are considered that have direct bearing on electromagnetic fields as potential causes for the various health endpoints under discussion.

This is particularly important with the case of electromagnetic fields, where the epidemiological evidence is, at best, very weak and the biological data are on the whole strongly unsupportive of carcinogenesis, or of any toxic effects at typical environmental exposures.

Despite its appearance of rigor, the report has serious logical flaws on several accounts.

1. The analysis relies on choice between two alternatives - "causal" or "not causal". But one of these alternatives is a straw-man argument. It is very difficult if not impossible to adduce sufficient evidence in support of a conclusion that associations are not causal.

A second straw-man argument is that biophysical theory claims that adverse health effects are impossible. Impossibility arguments are seldom possible in science, and they are virtually never used in analysis of risk data.

The use of straw-man arguments of this sort as alternative decision outcomes creates a bias that, in my view, negates the entire report.

2. The logic of the analysis is very unclear and probably incorrect.

A. The conclusions are incomprehensible. Statements such "it is more than 51% possible" that there is causation are never explained and their meaning escapes me entirely.

B. The report seems to use a kind of Bayesian approach, calculating the probability of a hypothesis (that EMF causes a variety of health problems) on the basis of given evidence. For example, the report speaks of the "prior

probability" that EMF causes cancer (and other diseases). But the authors do not consider the other probabilities that Bayesian logic demands, and their conclusions have no logical basis.

A logically correct analysis would require evaluation of biological data relevant to health endpoints, with consideration of their positive and negative predictive values. This is the approach taken by health agencies in weighing the evidence for a suspected risk. These considerations are nearly completely absent from the report. Instead, the authors cite a grab bag of biological studies. They cite reported biological effects that have no particular relevance to disease, and do not cite the negative results from studies that are relevant to hazard identification.

3. The panel of three reviewers is very poorly constituted. All three reviewers work in the same department, have published together, have similar professional specialties related to epidemiology and biostatistics. In particular, Neutra and Lee are co-authors of the miscarriage study that is the chief basis of the report's finding that EMF is likely to cause miscarriage.

Without intending any criticism of the individuals involved, a far broader committee is needed, particularly with includes biologists with expertise in in vitro and in vivo studies for carcinogen identification.

I hope that these comments are helpful. My work in preparing this analysis was not supported by an outside funding agency.

Sincerely yours,

Kenneth R. Foster
Professor

Raymond R. Neutra, M.D., Dr.P.H. Chief
Division of Environmental and Occupational
Disease Control
DEPARTMENT OF HEALTH SERVICES
1515 Clay Street, Suite 1701
Oakland, CA 94612

Re: Comments on the Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields (EMF) Draft Document of April, 2001.

Dear Dr. Neutra,

The following are our comments in regards to the above referenced document. We submit our comments in the form requested by you in your letter of July 9, 2001.

Page #	Table#	Line # or Comment # In Table	Comments
All		All	Overall the document is well written and the underlying thoughts are well developed and understandably expressed. However, we would like to have certain aspects and conclusions be presented in a way that avoids setting decision makers immediately on a certain track. Decision makers should be given the opportunity to see all aspects and burdens that the EMF issue places on society. We will explain this in the comments below.
1		17 to 34	In these two paragraphs the reader is presented with a cost-benefit analysis that compares the costs of EMF avoidance measures only with the costs of avoided deaths. Although there are also health costs other than the cost of lost lives (there are also survivors of EMF caused or promoted diseases with treatment costs that are significant), we know from the Power Grid and Land Use Policy computer model, that health costs are a magnitude lower than other costs of EMFs to society. For example, the statewide amount of devaluation of properties near overhead power lines is more than 10 times higher than the costs of lost lives. Also, one cannot just compare the construction costs of EMF mitigation measures with the benefit of saved lives. As we have seen from the computer models and as it was discussed before and during the development of the models, life cycle costs of EMF mitigation measures need to be taken into account in a cost-benefit analysis.
1		17 to 34	Continued… All these issues are lost in the two paragraphs of the Abstract and also in the main body of the document (including Tables 1 and 2), giving a decision maker an incomplete picture of the EMF issue for a cost-benefit analysis, setting him or her on a wrong track. ***Even a utilitarian would need to take all benefits from EMF mitigation into account and would need to compare these costs with the life cycle cost of the mitigation measure.*** It does not help that these issues are discussed on page 7, lines 1 to 15 of the document, when in the Abstract and in the Tables 1 and 2 these facts are not mentioned and not applied.
3		21 to 25	Same problem as described above.
3		50 to 55	Same problem as described above.
4		23 to 33	Same problem as described above.
5	1	UNIT COST	Live cycle costs need to be applied instead of construction costs.
5	1	STATEW.	As stated above, only comparing EMF mitigation costs with avoided deaths

Page #	Table#	Line # or Comment # In Table	Comments
		DEATHS….	results in an incorrect picture. All benefits, not only health benefits, belong here.
6	2	Cost…	Live cycle costs need to be applied instead of construction costs.
6	2	…Deaths to Avoid…	As stated above, only comparing EMF mitigation costs with avoided deaths results in an incorrect picture. All benefits, not only health benefits, belong here.
6	2	All	For a better understanding, the rows "Statewide costs…" and "Statewide survey costs" should be placed directly under the row "Number of affected schools" and before "Statewide total costs"
7		16 to 48	Again, the cost-benefit analysis focuses on deaths avoided by EMF mitigation measures. ***All benefits*** need to be compared with ***life cycle costs*** of such measures.

We suggest that the document be reworked so that the issues discussed in this letter are made more transparent to a decision maker and the tables contain all benefits compared to life cycle costs of mitigation measures.

Sincerely,

Peter Frech
Executive Director
Citizens Concerned about EMFs
P.O. Box 120
San Ramon, CA 94583

Dear Mr. Collins:

I have some questions about DHS's recently released report on Dangers of EMF which I hope the Department can address.

I don't know what the "conclusions" mean. To wit:

"It is "more than 50% possible" that EMFs at home or at work could cause a very small increased lifetime risk of childhood leukemia, adult brain cancer, and amyotrophic lateral sclerosis (ALS, Lou Gehrig's Disease). As this phrase implies, there is a chance that EMFs have no effect at all.

It is "more than 50% possible" that EMFs at home or at work could cause a 5-10% added risk of miscarriage, and again, as this phrase implies, there is a chance that EMFs have no effect at all.

It is "10- 50 % possible" that residential or occupational EMFs could be responsible for a small increased lifetime risk of male breast cancer, childhood brain cancer, suicide, Alzheimer's disease, or sudden cardiac death. As this phrase implies, there is a chance that EMFs have no effect at all.

It is "very unlikely (2- 10% possible) but not impossible," that residential or occupational EMFs could be responsible for even a small fraction of birth defects, low birth weight, neonatal deaths, or cancer generally."

What does "more than 50% possible" mean? Something other than "more than 50% probable". How is such a figure arrived at? What does "possibility" even mean? How do you define it? How is it different from "probability"?

Is this the result of some sort of Bayesian calculation? If so, what were the probabilities used to do the calculation? What were the prior probabilities? Or was the term chosen BECAUSE of its very lack of definition?

The question regulators and the public need to have answered is: What is the best estimate of the factor by which the incidence of (for example) childhood leukemia is increased due to exposure to EMF? If that factor is greater than 1, then they'd need to know the best estimate of what it would be given various amelioration methods. Then the politicians could weigh lives and cost and alternative uses for money.

What do "possibilities" tell us that is helpful for making these needed decisions?

Or were these "possibilities" just the uncalculated gut feelings of your 3 experts. Would a poll of the American public give a valid "80% possibility" of the existence of God?

Several major U.S. scientific organizations have asserted that there is no detectable deleterious effect of EMF on humans. How do you account for the difference between your findings and theirs? Some (especially foreign) organizations have felt there is a significant danger. Same question.

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September 10, 2001

Mr. Jack Collins
California EMF Project
Department of Health Services
Division of Environmental and Occupational Disease Control
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By Email: jcollins@dhs.ca.gov

Dear Mr. Collins:

Thank you for the opportunity to review the 3rd draft of the Electric and Magnetic Field (EMF) Risk Evaluation. It is a very substantial document and clearly exhibits the great deal of thought and careful work that has gone into its preparation. In a number of respects it breaks new ground in the arena of public documents intended to inform the public and policy-makers about the hazards, if any, of EMFs. We have already discussed some of the innovations with DHS staff during the preparation of the guidelines for conducting the evaluation. But it is perhaps worth reiterating that it is a valuable and unique contribution that the document 1) identifies differences in different scientific disciplinary approaches to the issues and considers with care cross-disciplinary implications; 2) considers that the users of the evaluation may well bring different ethical perspectives to the issues and care most about different aspects of the evaluation; 3) creates separate descriptions for pro and con interpretations of evidence to better illuminate both the evidence and the considerations that go into its evaluation; and 4) provides a variety of windows through which the reader can make his or her own interpretation of uncertainty in the judgements involved including both quantitative and qualitative descriptions by the evaluators, and the opportunity to observe the differences between the judgements of the individual evaluators and their explanations of how they formed their judgements. Also valuable are 1) the historical review which provides an important context for the evaluation, 2) detailed consideration of EMF exposures as a possible mixture of sources of physiological harm, and 3) the attempt, although we do not consider it fully successful, to explain the general approach of probabilistic updating (what might appropriately be called an informal Bayesian approach). We will discuss our concerns about the latter shortly. Two other novelties are extremely important, but create some problems for the way the document is written. One is that it presents new scientific information on possible EMF effects on miscarriages. The second is that the mode of analysis for the evaluation is sufficiently novel that it has led to some novelty in judgement: the analysis goes beyond providing simply another version of the standard review of the literature with perhaps some differences in interpretation. So we will now turn to our general concerns, which are centered on the treatment of those novelties. We will then, briefly, address the eight questions asked of reviewers and will finally make a few specific comments on the text.

The new study showing an apparent relationship between EMF exposure and miscarriages is clearly important and the document would be deficient not to include it prominently. However, new findings are different in character from the results of old studies which the scientific community has had more opportunity to review and to digest the implications of. The document acknowledges this difference explicitly in the history and in the measurement sections and implicitly by providing the relevant paper in an appendix. In our view, more needs to be done. The fact that there is a new information problem created by the appearance of the study (and other new work considered) should be stated at the beginning of the evaluation, along with the DHS choice of approach for dealing with it – namely to include it in the evidence considered by the evaluators (which is an appropriate choice) and to present the specific study in an appendix.

The finding that the evaluators' conclusions (posterior judgements) depend critically on their assessment of the weight to the various streams of evidence and less critically on their initial (prior) expectations is not itself new, but it is documented much more extensively than is usual for an evaluation document and it is important for

understanding the long-standing debates in the field. The finding is further illuminated by the differences between the answers developed for the California classification of confidence in causality and the IARC classification. The phrasing of a question affects the answer. Here again we believe that it would be better to state at the beginning that the evaluation has produced a more in-depth interpretation of the reasons for the persistence of the scientific debate, that that interpretation is a finding in itself, and that the evaluators happen to come down on a particular side – though not dogmatically so and without extreme overconfidence.

We would ourselves have preferred a somewhat different treatment of the weight ascribed to the biophysical arguments and to the experimental evidence, and instances of our preferences are noted below. With respect to the biophysical argument, we think it worth distinguishing between "laws of nature" and "theoretical modeling". If what is required for EMFs to cause health effects is really violations of the first or second laws of thermodynamics, we would consider this a very serious argument. But we agree with the evaluators, that the arguments have not yet met the test of showing that the limitation is not one of deficiencies in modeling, and the ongoing evolution of the model arguments seems to reinforce that conclusion. With respect to the experimental evidence, we would weigh somewhat more strongly the failure to find positive evidence and convincing mechanisms; there has been considerable work in this arena; but this is a judgement call that the evaluators were asked to make.

The descriptions of probabilistic up-dating are a considerable advance over the usual such discussion and for example offer new and good descriptions of how different kinds of evidence have different implications for different inferences. But we think serious problems remain. They include some fundamental definitional problems along with the general problem of communicating a technical mathematical representation to a group of people, not all of whom will understand or accept such representations. We think it would be helpful up front to explain that the evaluation was designed among other purposes to provide numbers for the decision-tree policy analysis, that such an analysis is best understood as following a utilitarian perspective, then to describe, briefly, what will be done with the numbers, and to offer alternative approaches including social for interpreting the results. Among the definitional problems – page references are noted below – are 1) the treatment of uncertainties in the individual expressions of confidence as illustrated, for instance in the graphs Table 1, pages 11-13. We do not see a clear definition of what the beautifully illustrated spread in values means. What would be the difference between a narrow distribution around 50% confidence and a broad one for instance? Would the difference reflect the evaluators willingness to argue for a particular value? Is it a spread reflecting an uncertainty in the prior or is it a judgement about how the evidence is to be read? Suppose that the evaluator has just two relatively strong, but contradictory sources of information, would that produce a broad or narrow range of confidence? How about many ambiguous results? Or very little pertinent information? And 2) another definitional issue is the use of confidence in the weight of evidence to "adjust" the number of health effects caused; we think a bit of further explanation, that you might make such an adjustment using a decision-theoretic utilitarian state of mind, would be appropriate.

Answers to questions:

1) As noted above we believe that it would be worth making clear the difference between "theoretical modeling" and "laws of nature". The biophysical argument is often presented as reflecting the constraints of the second law of thermodynamics and any evaluator should be strongly influenced by violations of that. The critical argument is that what is really at issue is how well the simplified models reflect the underlying biological situation; and in that respect we share the skepticism of the evaluators.
2) While we think the prior choices and their rationale are reasonable, we mostly want to note the following: it is an important innovation to disclose these for each evaluator; the final determinations are only moderately sensitive to these priors and that is a worthwhile finding; and that the critical sensitivity is the weight of evidence accorded to the various streams of information.
3) We would perhaps put a slightly higher weight on the failure, after considerable attention to come up with plausible models. But we regard our disagreement as still lying within the appropriate range for the evaluators to exercise their judgement.
4) And we have a similar view about the experimental studies. Though we would have put some of the evidence of biological effects up in the discussion of the "theoretical modeling".

5) Evaluating risks is a different exercise from constructing an epidemiological interpretation, and modest risks (as they appear in the environment) are the usual concern for risk evaluation. If epidemiological information is to be fully used in this enterprise, it should not be restricted to cases where there are large effects.

6) We agree with the arguments as presented, but want to note that they imply some general understandings about mechanisms; this suggests to us that mechanistic information, or the lack thereof, carries some weight.

7) While we think the basic arguments are presented quite fairly, indeed remarkably so, it might helpful to describe up front the basic conundrum: that there is a persistence of epidemiological findings but a persistent failure to find supporting evidence elsewhere.

8) The point of verbal descriptions is to provide an alternative to the mathematical representation: to be properly informative it will necessarily serve as a different sort of descriptor, conveying different informational value. Concern about the 50-51% overlap is only appropriate if the evaluators believe that they can make such a distinction, or that they know the balance of probability that well. Perhaps a bit more discussion of qualitative phrasing and why the numbers are not precise and do not reflect the exact nature of the judgements made would help more than trying to rephrase the categories.

A few specific page references

Page 1, line 8 – rephrase here (with the implication for the other points below) " As the phrase implies, there is *in the judgement of the evaluators also a substantial (significant)* chance that EMFs have no effect at all.
Page 4, line 26 this sentence should be revised and explained (see above)
Page 8, line 18 on – it would be nice to have an added sentence indicating the reason
Page 15, footnote – here this is not a fair representation of the Challenger situation – but the illustration is valuable and could be saved by a sentence that says that this is a simplified hypothetical interpretation of the Challenger situation.
Page 15-16, the section on uses is good, and should be stressed elsewhere
Page 17, line 22+ , as discussed above, this multiplication is problematic and should be discussed further
Page 18, line 9+ and here it is again with a different, aribitrary factor
Page 19 line 31, this sentence is fine, but we note that it does imply a concern with mechanistic and experimental streams of evidence.

We would be happy to discuss the main points in more detail and provide some more specific comments on the text if this is desired.

Sincerely,

Robert Goble
Research Professor

Dale Hattis
Research Professor

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Department of Environmental Health Sciences
Division of Occupational and Environmental Health

November 6, 2001

Raymond Neutra, M.D., Dr.P.H., Chief
Division of Environmental and Occupational Disease Control
1515 Clay St., Suite 1701
Oakland, CA 94612

Dear Dr. Neutra,

Thank you for asking me to review the "Electric and Magnetic Field (EMF) Risk Evaluation". Generally, it is a well-written and well-thought report that contributes greatly to the assessment of electric and magnetic fields, as well as to the practice of policy development in environmental health. I think that generally the Bayesian approach that you have developed is very consistent with how policy makers think about risk. More specifically, your approach provides a great deal of transparency, which should allow all who are concerned about this issue to completely understand the assumptions and the logic that is built into your risk evaluation framework. I would consider this to be an important breakthrough for environmental health policy. A next step would be to develop tools for synthesizing all of this into smaller, more comprehensible, bites for decision makers. Such an effort would need to engage either current or past decision makers in addition to scientists.

To address your specific questions:

1. I would agree that physics and biological models that suggest that residential and occupational levels of EMFs cannot be expected to produce biological effects should not cause you to have a "vanishingly small" prior degree of confidence in the results of epidemiology studies. The epidemiology studies should be judged on their merits.
2. The judgments that are given by the core reviewers seem reasonable. I would not wish to venture a personal judgment on EMFs. Generally, I have a strong a priori degree of confidence that a number of environmental/occupational agents are associated with cancer, and that individual agents probably have small attributable risk fractions in most instances.
3. I probably would place more weight on the fact that, to date, mechanistic explanations of how EMFs can cause biologic effects do not point to a convincing chain of pathology. However, it is important to recognize that in public health we almost never have proof of the mechanism of action for toxic substances, though we often have theories.
4. I do not understand the animal pathology literature well enough to comment on this point.

5. I agree that relative risks in the range of 1 to 2 should be taken seriously, absent evidence for confounding or bias. Well-conducted epidemiology studies can reliably estimate risks in this range. It is important that we factor such information into decision making in public health.
6. I agree that there should not be a concern for lack of specificity in the association of EMFs with subtypes of cancer. A number of known human carcinogens, e.g,, benzene, asbestos, and arsenic, cause several types of cancers.
7. I think you have done an adequate job in presenting the arguments for and against causality.
8. Here is a proposal for an alternative chart and language to describe it:

Confidence Range	Suggested alternative
> 90%	Strong evidence for causal relationship
61-90%	Strong evidence for probable relationship
41-60%	Limited/suggestive evidence of possible relationship
< =40%	Inadequate evidence whether or not there is a relationship

I like this framework for a number of reasons. First, if your confidence level is less than a coin toss, you are left with the option of either doing more research or moving on to other issues. Second, when your confidence is in the range of 50% I don't think it is wise or useful to split hairs over whether it is 49% or 51%. In my experience, issues that fall within that range can have risk management implications, even while more research is underway.

Thank you again for the opportunity to comment. Best of luck with this important effort.

Very truly yours,

Lynn R. Goldman, MD, MPH
Professor, Environmental Health

August 30, 2001

Mr. Jack Collins
California EMF Project
California Department of Health Services
1515 Clay Street, 17th Floor
Oakland CA 94612

Dear Mr. Collins,

I am writing in reply to Dr. R. R. Neutra's letter of July 9, 2001, requesting comment on Draft 3 of the EMF risk evaluation document. In the next several paragraphs I first will make several general comments; next, some specific ones; and finally, address the specific questions posed in Dr. Neutra's letter.

1. General Comments

Overall, the review reflects a great deal of thoughtful work and, particularly in its presentation of ranges of uncertainties, presents a rare and more balanced view of the complicated nature of the risks being presented. The statistical approach will be considered hard to understand by many, particularly the role of the a priori estimates, which get much discussion but which I'm not quite sure I fully understand.

This balanced view of a complicated situation does not always penetrate into the public statements, however; they read like boiler plate, and the condensed statements in places like section 21.0 (pp 301-304) and Table 21.1's "IARC Class" and "Confidence" columns don't seem to me to summarize the "Degree of Confidence" graphic. Compare, for instance, the only (R-2) estimate (for air pollution) and R-1's assessments for Childhood or adult leukemia. Or the ALS and Suicide estimates. The latter suffer from being broad ranging estimates that happen to be centered on opposite sides of a boundary. But that's enough to put them on opposite sides of the dichotomy.

In addition, I found that in spite of the large section on methodology, I had to dig carefully into the first section of the Executive Summary (p. 6) to learn who did what, the role and identity of the reviewers (otherwise kept scrupulously anonymous), the rationale for using these reviewers, etc. Normally, information in an Executive Summary is backed by more extensive information in the main body of the report.

After having dug the "who's who" information out and thinking about the process and the report, I was left with the impression that this was an impressive piece of work by three individuals in DHS, all of whom were basically epidemiologists. Had I not had outside knowledge of these three—all good scientists—I would have wanted more information about each of their qualifications than was on p. 6 ll 43-45. No cv's? The document spends relatively little time on any of the non-epidemiological research, though there are a great many references available. I'm not sure I (as a non-epidemiologist) would have come to different conclusions about the value of the non-epi work than the document does, but I probably would have either spent a significantly greater time on it or would have explicitly deferred to the NIEHS or other analyses.

There is no indication of the contributions or broadening insight from the more heterogeneous scientific advisory panel or other members of the staff of DHS, some of whom presumably did at least part of the basic work. I found myself thinking that, irritating as it might be, the Federal Register practice of summarizing external comments and indicating what changes they have or haven't produced, along with a rationale, could have been used at various points to indicate where the panel and DHS staff—as well as where the various external comment processes for Drafts 3 and 4—had contributed. There is also the dual role that at least Neutra and Lee are playing, since they are coauthors of some of the studies, including some of the newer ones appended to the report. There is an impression that they are critiquing their own work. Del Pizzo is the only one of the three with at least some sort of separation between essentially all of the work cited and this analysis.

All of these reasons make the difficulty in finding out who "the reviewers" were all the more irritating. More importantly, they weaken the possible impact of the study by making it seem as though it is the opinion of three epidemiologists from CHS.

2. Specific Comments:

I was halfway through the book before I realized that, quite properly, the RR charts had a vertical scale that is logarithmic, making increases and decreases spatially equivalent. They also had horizontal lines that indicated specific levels of RR, not (as I had begun to assume) mean/SD for the pool of experiments under discussion. It might help to flag these aspects of the charts somewhere, though the discussion would get missed by many. Maybe it's there, and I missed it.

There are several places where ideas are not apparently treated consistently between chapters. This idea is not carried forth elsewhere in the paper, though probably it could be. Sedentary activities and work stress associated with VDTs are discussed as possible confounders of birth defects in Ch. 14, but not of miscarriages in Ch. 13.

Certain ideas are introduced, but not followed up or placed on the research agendas. Table 8.2.7 introduces the idea of a sigmoidal dose-response that is also seen in many cell experiments, though hardly all. Shocks of various types are discussed and dismissed, though not on the basis of data, in Ch. 15; personal communication I have had with people who work with power company line workers indicate that shocks are common, considered one of the hardships of the job and to some extent badges of honor, and are generally unreported unless requiring immediate medical attention. There ought to be data on this somewhere. In general, electrical workers are disproportionately represented as subjects in the pool of epi studies. But I didn't see any comment on this.

While there was comment on the quality and relative reliability of various studies, no explicit weighting was apparently done in the analysis in the various chapters. Presumably the reviewers did some tacit weighting in arriving at their conclusions. While meta-analyses generally don't feel there is enough evidence for explicit weighting in their calculations, it would be interesting to know whether the reviewers were relatively consistent concerning the weights they gave the various studies.

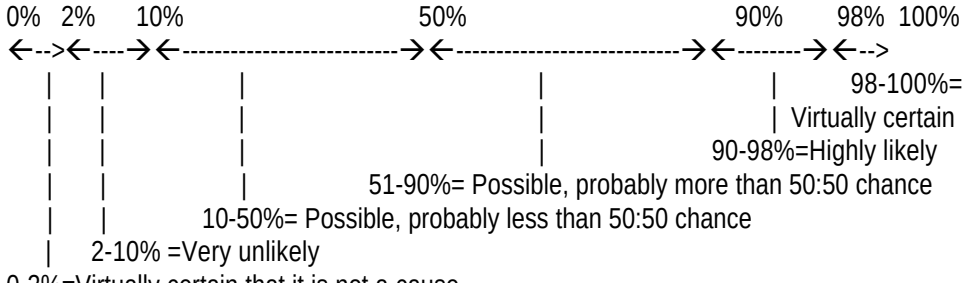
3. Specific Questions in Letter:

1. Models based on physics are generally simplified. They can be quite useful if the simplifications do not throw out crucial aspects of the situation and can be quite misleading if these aspects are overlooked. The only way one can decide which is the case is to look at the data that one trusts. Data that is good—good experiments, reproduced, etc., casts doubt on the model. Data that is marginal may become more suspect if the model contradicts it. Basically, I support the general idea used in the study.
2. I tend to come at the prior estimates in a way similar to reviewers 1 and 2, though I would caution reviewer 1 that while no specific repair mechanism for EM fields would have evolved over time, no EM-specific damage mechanism would have evolved, either, making existing repair mechanisms for existing types of damage at least partially relevant. The stress protein work has some relevance here; the issue is the degree to which EM adds to other stresses to the point where EM tips the balance of an organism becoming unable to ignore the sum of the effects of its environment.
3. I agree with this point.
4. I would have given a bit more weight to some of the studies of behavior and of gene expression. Not much more, given the inconsistency between labs and the gap between in vitro/animal and human studies, but some. Epi got more discussion and more weight, as noted above, than other types of studies.
5. Some of this is a technical discussion among epidemiologists, but I think that the overall consistency of a significant number of good studies should be considered worthy of notice.
6. This was an interesting point and worthy of further discussion. I do not discard it out of hand. Are there analogous situations in chemical toxicology or malnutrition?
7. As noted above, the arguments are not always consistent between chapters, presumably reflecting the assignment of various parts to various people and perhaps less review by the three reviewers and by

others of the preliminary work. (Did the three review each of the sets of statements? If so, was this done before or after they formed their individual conclusions or afterwards? Different answers to these two questions could indicate a tendency toward different outcomes. Concern about the apparently small number of people involved—the 3 reviewers—shows up here.)

8. ANY set of phrases is going to be a problem. Prominently displaying the table of confidence range vs. phrase, supplementing it with a graphical view (divided bar or line), etc. is the only way to reduce the problem. You won't eliminate it. Suggestion below may or may not be better for a non-technical reader: Emphasize that boundaries are arbitrary and that position along 10-50 and 51-90 ranges reflects differences in degree of likelihood:

Level of Confidence:



Sincerely yours,

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A Critique of Draft 3 of "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMF) from Power Lines, Internal Wiring, Electrical Occupations, and Appliances" by the California EMF Program of the California Department of Health Services (DHS)

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DISCLOSURE AND DISCLAIMER

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PREFACE

In the Fall of 2000, following the publication of the pooled analysis of magnetic fields, wire codes, and childhood leukemia by Greenland, Sheppard, Kaune, Poole and Kelsh (2000), Prof. Charles Poole (of the University of North Carolina Department of Epidemiology) and I were enlisted by Dr. Raymond Neutra of the California EMF Program of the Department of Health Services (DHS) to come to the Program offices and comment critically on the methods he and his colleagues were using to evaluate the EMF literature. Both Dr. Poole and I were extremely critical and pointed out numerous statistical and logical errors in those methods. The draft (Draft 3) critiqued herein was thus prepared following some sharp verbal input from me, as well as from Dr. Poole.

Following this interaction with DHS, I heard nothing more until Summer 2001, when various colleagues (none from DHS, the Electric Power Research Institute (EPRI), or the utility industry) brought Draft 3 to my attention. Although this draft had removed the erroneous formal Bayesian calculations present in the DHS materials of the previous year, most of the other errors pointed out by Poole and me remained. Nonetheless, because I was preoccupied with many other matters, I did not intend to file public comments on Draft 3. I did however describe and criticize some of its major errors in public presentations before statistical and epidemiologic audiences, and before a workshop on selection bias in EMF studies held by EPRI in July 2001. Soon after that Dr. Jack Sahl (formerly the epidemiologist for Southern California Edison (SCE), now a private consultant) asked if I would be willing to write down and submit comments on Draft 3 to DHS, with my time paid for by a consortium of California electric utility companies. I agreed, given the stipulation that those sponsors and their affiliates would have no input or access to my product until it had been submitted to DHS and thus become a public document.

My current probability ("confidence") that residential magnetic fields can cause childhood leukemia is about 50%, a little below the 55% given by DHS Reviewer 2 (Draft 3, p. 103, line 18) and far below the 99% given by DHS Reviewer 1 (who is "virtually certain": see Draft 3, p. 101, line 21). My probability is however above that of all my colleagues at the July 2001 EPRI workshop and all other EMF researchers I have queried (none of whom have answered above 40% so far).

Residential magnetic fields either do or do not cause childhood leukemia, so from my perspective there is a 50% chance that Reviewer 1 believes in the correct hypothesis. The point of my sharp criticisms of Draft 3 is that the certainty of the DHS reviewers is not grounded in sound reasoning from the available data. From my perspective, everyone who (like Reviewer 1) is convinced that magnetic fields cause childhood leukemia also has a 50% chance of believing in the correct hypothesis, even if they became convinced based on tarot reading or voices in their head; it does not matter that the methods they used to arrive at their decision are entirely untrustworthy for arriving at correct conclusions. I focus here on whether the evaluation methods used by the DHS reviewers to form their confidence are trustworthy for policy formation, in the sense of avoiding scientifically unsound extremes of confidence. I will argue that those methods are not sufficiently trustworthy, as evidenced by the extreme certainty of Reviewer 1 regarding childhood leukemia.

For the record, I have served as a consultant to and collaborator with health researchers at EPRI and SCE, so I have ties to the utility industry beyond the funding of this critique (though I own no stock in the industry). On the other hand, I have also served as an expert witness for the plaintiffs in a large-scale lawsuit against Pacific Gas & Electric regarding hexavalent chromium contamination of communities from PG&E cooling towers, and have served as an expert for the California State Attorney General's office regarding product-risk assessment, so I have some ties to public-interest actions as well.

SUMMARY

Draft 3 presents judgments by three reviewers who work for the DHS. These reviewers conclude it is "more than 50% possible" that EMFs at home or work could cause a very small increased risk of childhood leukemia (page 1, line 7). I have examined in detail the reasoning and data used to reach this judgment, as presented on pages 1-109 of Draft 3. I have concluded that the reasoning suffers from serious errors of logic and statistics, that there is repeated counting of the same data and subjective priors in the formation of conclusions, and that certain key data are not accounted for properly. There is also evidence of bias in the reasoning of the DHS reviewers away from the null (against the hypothesis of no effect), toward concluding there is an effect. This general bias is evidenced in the summary tables in Chapter 8 of Draft 3, which display a tendency to misinterpret negative

evidence (favoring no effect) as neutral or positive, to misinterpret neutral evidence as positive (favoring an effect), to double-count only positive evidence and opinions, and to ignore literature aspects that would call into question the validity of the epidemiologic studies.

These problems have led to overstatements of confidence about EMF effect in light of available data, and render much of Draft 3 invalid. DHS Reviewer 1 displayed so many error and gaps in his knowledge of statistics, epidemiologic methods, and the underlying data sources, along with fallacies of reasoning, that his evaluations should be discarded. The evaluations of DHS Reviewer 2 are not as misleading, though they need revision in light of several important considerations, especially much more consideration of possible participation biases across studies. Reviewer 3 fell between Reviewers 1 and 2 in overconfidence. Although a revised assessment by Reviewer 2 may be adequate for informal purposes, production of an accurate risk assessment would require much more expertise in epidemiologic and Bayesian statistics than evidenced by the three DHS reviewers, as well as use of that expertise to execute a detailed analysis of sources of bias in the epidemiologic studies.

OUTLINE

The remainder of this critique is organized as follows: First, I give a general summary critique of the basis and methods for the Draft 3 evaluation, indicate what a more valid risk assessment would look like, and speculate on the results it would produce. The core criticisms are that Draft 3 focuses on subjective elicitation of posterior opinions from three nonrepresentative researchers, rather than on logically sound deduction of conclusions from representative sets of plausible premises; that several major statistical errors and logical fallacies are built into the DHS evaluation methods; and that major sources of uncertainty are not accounted for properly. Second, I describe in detail some crucial errors in reasoning and statistics, as well as gaps in literature considerations, in order of their appearance in Draft 3. Third, I give a set of technical appendices to further show how these errors and gaps lead to overstating the strength of posterior certainty (confidence) about the hypotheses (effects) at issue.

GENERAL SUMMARY CRITIQUE

A fundamentally misleading aspect of Draft 3 is its implication that the "semi-quantitative posterior degrees of confidence" (p. 22, lines 65-66) it offers as conclusions are founded on "using Bayes formula as a heuristic" (p.22, lines 56-57). Unfortunately, the major messages and methods of Lindley (2000) (cited on lines 31 and 53 of p. 22) and other leading Bayesians (e.g., DeFinetti, 1974; Good, 1983) have been ignored completely in the process described in Draft 3; in addition, the methods used in Draft 3 are replete with elementary errors of statistics and logic. The consequence is that the conclusions of Draft 3 have no justification in any statistical or logical sense, Bayesian or otherwise, and hence are not valid inputs for a decision-analysis model.

As has been carefully documented in cognitive research (e.g., Kahneman et al., 1982; Morgan and Henrion, 1990, Ch. 6; Piatelli-Palmarini, 1994), intuitive and informal reasoning about evidence and probabilities of hypotheses is exceptionally poor, even in simpler settings than the present one, and even when done by experts. A crucial role of Bayesian analysis is to ensure that any conclusions (posterior assessments) follow from any premises (prior and likelihood assessments) in conformance with the basic rules of deductive logic and the universally accepted axioms of probability (see citations above, as well as Howson and Urbach, 1993, and Greenland, 1998). As will be documented below, Draft 3 repeatedly fails to check the logical connection of its priors, likelihoods and data to its conclusions, especially in its evaluation of biases; as a consequence, it commits a number of fallacies which exaggerate its confidence in its conclusions. Draft 3 attempts to rationalize its failure to check its reasoning quantitatively by claiming its goal in invoking Bayes' theorem is to make its "assumptions more explicit" (p. 21, lines 34-37). While making assumptions explicit is an essential part of any valid risk analysis, it is not sufficient, and its value is almost nullified if the reasoning from those assumptions is fallacious, as in the draft.

As a separate issue, the three priors elicited and used in Draft 3 are by themselves of doubtful value to EMF risk assessment because they do not satisfy basic conditions for general credibility or sensitivity analysis (that is, they do not encompass a representative range of expertise and viewpoints of researchers in this topic area; see

Good, 1983 and Morgan and Henrion, 1990, sec. 7.7). First, the three priors are highly interdependent, as they come from three reviewers in the same department, at least two of whom have worked together extensively for years on this topic. Second, the three priors are highly nonrepresentative. Reviewers 1 and 2 are generally regarded as falling to the pro-effect side of the controversy; furthermore, Reviewers 2 and 3 are epidemiologists and Reviewer 1 is a physicist by formal training and qualification. No priors were elicited from scientists who have research qualifications in observational statistical theory or in bioassay, and no priors were elicited from more moderate (let alone skeptical) researchers in this topic area.

A valid scientific assessment explains how its conclusions can be logically deduced from plausible background assumptions and data likelihoods (cf. Maclure, 1998). Draft 3 does not do this; instead it uses "posterior elicitation" (p. 24, lines 9-10) to derive its conclusions. Such elicitations could have been useful for back-calculating to priors by reverse-Bayes (Good, 1983), provided credible data likelihoods were used; that calculation would have made clear that the conclusions of Reviewer 1 were largely derived from biased reasoning and from statistical errors. Posterior elicitation is not, however, a valid method for scientific risk assessment; because it lacks explicit deductive derivation, it is especially vulnerable to cognitive bias (Piattelli-Palmarini, 1994).

Another way to see the underlying structural inadequacy of Draft 3 is through cognitive-systems theory for causal analysis (Pearl, 2000). Valid learning and decision making, including risk assessment, have two basic requirements. The first requirement is a reasonably accurate model (priors, assumptions) for the processes generating the input data, where "reasonably accurate" implies that, among other things, all important effects (including bias sources) are represented by functions flexible enough to approximate key properties of those effects (dose-response shapes, etc.). The second requirement is accurate computation (valid deduction) under that model. As will be documented below, Draft 3 fails both requirements. For example, the implicit model (assumptions) used by Draft 3 for potential socioeconomic effects on the epidemiologic data (via confounding and selection bias) is incognizant of and in conflict with existing data. As another example, Draft 3 incorrectly assumes that unknown but possible systematic errors should not reduce posterior certainties.

Intelligibility Issues.

On page 22, lines 53-57 of Draft 3, the authors attempt to excuse their failure to carry out a logically sound analysis by claiming such an analysis "requires complicated and probably unintelligible modeling and cannot be done with a simple calculator and Bayes equation." I have no doubt that this is their honest reason, and it amounts to admission of their inability to carry out a valid risk assessment.

First, failure to carry out valid analyses because they "cannot be done with a simple calculator" is an appalling excuse in the 21st century. Multiple logistic regression cannot be done with a simple calculator, and yet has been the standard statistical method for case-control analysis for some 20 years. Today anyone can buy a giga-Hertz speed computer for under $1,000, and perform a Bayesian analysis with software downloaded from the World Wide Web (see the WinBUGS website), or with ordinary SAS procedures via a few lines of documented SAS program code (also available on the Web; see Witte et al., 2000). Even if one is loathe to employ such software or methods, one can easily employ Monte-Carlo risk (sensitivity) analyses (Morgan and Henrion, 1990, sec. 8.5), which with suitable care can approximate Bayesian results (Greenland, 2001b). An example of such an analysis is given in the Appendix.

Second, complexity and intelligibility of modeling depend entirely on the understanding and explanatory skills of the analyst; the authors' comments document their profound misunderstanding of the very methodology they purport to be "using as a heuristic." Valid scientific and policy use of Bayes' theorem demands that the inputs to the formula (both the prior model and the likelihood model) encompass all the major sources of uncertainty in explicit form. If there are many sources of uncertainty, as in this subject, those sources must be represented by many parameters, and the model will be large, <u>as it should be</u> given the complexity of the topic. Large size in no way precludes intelligibility for competent analysts: Large models can be modularized so that each source of uncertainty is given its own intelligible and manageably simple submodel.

On p. 71, lines 50-53, the authors state that "our stakeholders made clear at the outset that we should not rely on a method that was not accessible for criticism to most readers." As shown below, Draft 3 is certainly accessible

for criticism. Nonetheless, the stakeholders and DHS should ask whether they would prefer an accessible but highly misleading assessment, as given in Draft 3, to a more difficult but less misleading assessment. I will argue below that the latter need not be inaccessible for criticism, even though it might be beyond the technical skill of the stakeholders and DHS reviewers to conduct. (As an analogy, we can all criticize the Windows™ operating system by documenting how poorly it responds to inputs, even though few of us could read the code, let alone create an alternative.)

By insisting on use of a risk-assessment process so simple that anyone can understand it without concerted technical effort, one virtually guarantees that the conclusions will be distorted by many oversimplifications. An example of the impact of such oversimplification is given below, in my criticism of p. 16, lines 23-25 of Draft 3, which derives figures in part from a DHS-funded analysis by Wartenburg (2001). That analysis used an untenably inflexible and oversimplified (one-parameter) log-linear model for the dependence of childhood-leukemia risk on magnetic fields; as a consequence, it more than doubled the attributable-fraction estimate from magnetic-field data over what it would have been under a realistic, flexible model. On top of this failing, Wartenberg (2001) labeled this analysis a "risk assessment," even though he provided no uncertainty assessment or sensitivity analysis of his result; consideration of just a few reasonable threshold models would have revealed the extraordinary sensitivity of his estimate to his modeling assumptions.

What Would a More Valid Risk Assessment Look Like and Produce?

A more valid assessment would at the very least correct the statistical errors and logical fallacies described below. In particular, it would discard the input of Reviewer 1, which is replete with upward bias and methodologic fallacies; it would take proper account of observed relations of socioeconomic (SES) indicators to participation and to disease; it would remove the "consistency" analyses from the list of causal considerations; it would allow for the certainty reduction that should follow from our ignorance about the source, size, and pattern of systematic errors; and it would replace the incorrect dose-response and attributable-fraction analyses of Chapter 20 (used in Chapter 8) with analyses that take proper account of model uncertainty. The resulting assessments would definitely appear much less certain that the evaluations given by Reviewers 1 and 3; they would probably appear

somewhat less certain than the more cautious evaluation by Reviewer 2, though perhaps not to a large degree. In any case, to provide a trustworthy basis for policy, extensive correction of the entire evaluation is needed, followed by external review by qualified experts in observational statistics and EMF epidemiology.

SPECIFIC TEXT CRITICISMS

p. 14, lines 39-46:

Here, Reviewer 1 takes the independence principle (product rule) of uncertainty assessment and applies it selectively and incorrectly in a manner that upwardly biases his certainty of EMF effects.

Independence is an assumption made by an assessor, analyst, or reviewer. Assertions about prior independence or dependence can vary widely across reviewers, and that variation can have strong impacts on conclusions. Nonetheless, once independence of statements is asserted, it implies that multiplication of the probabilities of the statements will yield the probability that all the statements are true (conjunction of the statements).

Reviewer 1, in footnote 1 on p. 14, cites a correct application of this rule to the Challenger space-shuttle disaster: The probabilities of six statements of the form “seal no. k will not fail” for k=1 to 6 are multiplied together to give the probability that all the statements are correct, i.e., the probability of the statement “no seal will fail”, under the (crucial) assumption that the six statements are independent. But independence is a reasonable assumption in this example only if one makes the probabilities depend on temperature and any other shared determinants of seal failure, a point not seen by Reviewer 1.

Now consider how the same rule should be applied to the situation described by Reviewer 1 on page 22, lines 43-46: Here we have the statement “the association between EMFs and childhood leukemia is not reasonably explained by chance, bias, or confounding.” This statement logically implies the conjunction of three statements of the form “the association between EMFs and childhood leukemia is not reasonably explained by X,” where X is

"chance" (statement 1), "bias" (statement 2), or "confounding" (statement 3). If the probabilities we assign to these three statements are Q_1, Q_2, and Q_3 respectively, AND we assume that the three statements are independent (an incorrect assumption, as discussed below), then the probability of them all being true (their conjunction) should be the product $Q_1Q_2Q_3$. As in the Challenger example, even if the individual statement probabilities are high, their product will not be so high (only 73% even if each Q_k is 90%), and so one should not be so certain that their conjunction is correct.

Reviewer 1's statement is even less certain than the rule indicates, however, for two major reasons: First, the real statement of scientific importance here is not whether one of chance, bias, or confounding alone could explain the association; rather, it is whether any combination (alone, two, or all three sources of error together) could explain the association. That is, the statement "the association is not explained by chance, bias, confounding, or some combination" is the relevant scientific hypothesis (statement) for Reviewers 1's line of reasoning (and is presumably the one he meant in lines 43-46). This statement is a logically more restrictive (less easily satisfied) hypothesis than the statement "the association is explained by one of chance, bias, or confounding alone," which is the conjunction of the three statements given earlier. Logically, this means that the probability assigned to the relevant statement should be strictly less than that assigned to the conjunction (which would be the product $Q_1Q_2Q_3$, assuming independence) (Howson and Urbach, 1993).

The second factor that should further reduce the probability of Reviewer 1's statement is that the three sources of error listed (chance, bias, confounding) are dependent in ways that further reduce the certainty of Reviewer 1's statement below that of the certainty indicated by the product rule (which assumes no dependence). One source of dependence is that the extent of both selection bias and confounding by a possible risk factor depends critically on the strength of the factor's association with the exposures of interest (here, wire codes and magnetic fields). In other words, selection bias and confounding are not independent explanations because they share important unknown component parameters that determine their sizes (the parameters for the association of factors with the exposure). The fact that the resulting dependence is unknown should reduce certainty about the source of the observed EMF-disease association.

p. 14, lines 50-54:

Reviewer 1 here asserts that "evidence of an association between EMF and childhood brain cancer obtained in the course of a study, which also examined the association with childhood leukemia, may have been affected the some the of the same biases. However, they would not be affected by the same confounders, because the two illnesses have different risk factors." Given that so little is known about the risk factors or etiology for either childhood brain cancer and childhood leukemia, one wonders how Reviewer 1 or anyone else could so confidently assert that the two illnesses share no risk factors. There is no data basis for this assertion, and the statement is a classic example of turning ignorance into knowledge: Absence of knowledge about shared risk factors in no way provides knowledge of absence of shared risk factors. There is at the present time insufficient data (even less data than for magnetic fields) to rule out or even seriously question the possibility of shared risk factors related to environmental, pharmacologic, or dietary/nutritional exposures (including in-utero exposures). This remains so even if other factors have been found to affect risk for one disease but not the other.

Merely allowing for the possibility of shared risk factors would increase the probability of shared confounding, because the shared risk factor would become a confounder for both diseases if it is associated with magnetic fields. Similar allowance for other shared sources of bias, as well as shared confounding, would increase the correlation (both prior and posterior) of the two effect parameters (those for the effect of magnetic fields on childhood leukemia and for the effect of magnetic fields on childhood brain cancer).

p. 16, lines 23-25:

Regarding the population attributable fraction for magnetic fields and childhood leukemia, Draft 3 here claims that "Greenland et al. (2000) and Wartenberg (2000) [actually 2001] estimated that residential EMF exposure might account for 0% to 12% of childhood leukemia, with a mid-point of 4%." This statement is incorrect. Greenland et al. (2000) gave an estimate of –2% (protective) to 8% with a midpoint of 3%, based on direct modeling of dose-response using the original data from eleven studies. The attributable-fraction estimate of 11% from spot measurements in Wartenberg (2001) is based on a grossly oversimplified model that upwardly biases the

estimates relative to the best-fitting models for the pooled data. Specifically, Wartenberg used published data summaries, not original data, and assumed a one-parameter log-linear model for the effect of magnetic fields across the entire range of exposure. Use of such a rigid, simple model has no justification in either theory or data; in fact the original data show no association below 2 mG, where 90% of the U.S. population resides (see Tables 5 and 6 of Greenland et al., 2000). Wartenberg's estimate is grossly inflated because his model misspecification resulted in small excess risks getting applied against the large population percentage below 2 mG.

p. 16, lines 25-27:

Draft 3 here states that "A reanalysis of the Greenland data, supplemented by the UK (1998) study ... narrowed the confidence interval but concurred with the 4% mid-range estimate." No supporting calculations were provided. From Chapter 20 of Draft 3, it appears that the calculations used did not follow the principles described in Greenland (2001a) (which derives and justifies the interval given by Greenland et al. (2000)): The Draft 3 calculations failed to properly account for the uncertainty in the dose-response form and the population exposure distribution, and hence produced an invalidly narrow interval. As will be discussed below, it is also apparent from Chapter 20 that the dose-response estimation method used for the calculation contained other errors as well.

p. 22, lines 31 and 53:

The Lindley (2000) article appears to not be in the references.

p. 22, lines 67-75:

Draft 3 here gives a brief description of the decision-analysis models that are destined to employ the "posterior degrees of confidence" from this report. While this description lacks enough detail to allow a full critique, the description given (along with the failure of the authors to grasp and employ valid methods for deriving "posterior confidences") strongly suggests that the decision models are also invalid when evaluated against long-

established quantitative decision-theoretic methodology (e.g., as in DeGroot, 1970; Cox and Hinkley, 1974, Ch. 10). Like the classic formal decision theories, even semi-formal practical decision theories require development of fully articulated posterior distributions as inputs (along with cost functions) for computing expected costs or benefits (e.g., see Morgan and Henrion, 1990). It appears that, instead of employing such distributions (which would supply probabilities of effects at each magnetic-field level), the EMF program's decision models use only the "posterior degree of confidence" that the 2 mG time-weighted-average relative-risk estimate is causal. A model based on such grossly oversimplified inputs could not begin to approximate a valid decision function, especially in light of the broad dose-response possibilities for magnetic-field effects (see Fig. 1 of Greenland et al., 2000).

p. 24, lines 29-32:

The use of 95th and 1st U.S. EMF percentiles to elicit priors is misleading for a number of reasons. First, from a biophysical perspective these percentiles are utterly irrelevant in themselves, as they refer to no particular level of exposure that could be related to any bioassay data or biologically-based priors. In effect, the elicitation is designed to ensure any such background is ignored.

Second, the use of just one arbitrary relative risk and two points for elicitation ignores the important structural information that exposure accumulates along a broad continuum. A priori, nothing was known about dose-response (and very little is known today, other than that EMF-leukemia effects, <u>if any exist</u>, are probably monotone along the relevant axes); and many response curves that begin to rise only well above the 95th percentile will produce population attributable fractions greater than many curves that begin to rise at much lower doses (in fact, the existing epidemiologic data suggest that any effects of magnetic fields on childhood leukemia are confined above the 95th U.S. percentile; see Greenland et al., 2000, Tables 5 and 6, and the UKCC study results).

Third, given that it is California policy at issue here, one should question why the U.S. percentiles were chosen (given that percentiles were to be used, incorrectly) when it is known that residential EMF distributions vary widely across states (as can be seen in comparing controls across U.S. case-control studies).

Many types of prior construction allow direct linkage to biologically relevant measurements and to dose-response priors. For example, one could specify a prior for the average effect of one increment of exposure (e.g., the relative risk from each 2 mG increase in the etiologically relevant but unknown field summary measure), then specify a prior for the deviation of these effects from the average; one could even adopt a semi-Bayes approach that would not require one to specify more than the qualitative structure and spread of the prior (e.g., see Rothman and Greenland, 1998, p. 431-432). More simply, one could just elicit correlated priors for a sequence of incremental relative risks (e.g., for 2 vs. 0 mG and 4 vs. 2 mG using a bivariate lognormal prior). The issue of uncertainty about the relevant EMF summary exposure could be subsumed under the prior for measurement errors (as shown below, the effects of the latter errors are accounted for incorrectly in Draft 3).

Chapters 3-6:

I am not an expert on physics, power engineering, biophysics, or bioassay. Therefore, I read these chapters as background only, and offer no critical review of them. Two minor tabular errors are noted below.

Table 3.1.1:

"X" was misprinted in my version of this table

Table 3.1.2:

The heading fails to state the table entry measurement units.

Continuation of critique (Ch. 7 and 8):

Table 7.1 and Section 7.2:

This table gives a list of questions used to guide the Draft 3 versions of Hill's (1965) considerations for evaluating causality. It is not noted here (but should be) that Hill, as well as many later authors, had many reservations and criticisms regarding these considerations, and later authors suggested modifications to produce considerations more relevant to scientific deduction (see Rothman and Greenland, 1998, Ch. 2 for a review). None of these modifications is incorporated here.

A major problem is that the explanations in most of these questions are cast in an all-or-none (qualitative) form, which biases the assessment against alternative explanations, and (like the other errors in this Draft) leads to overstatement of posterior certainty about EMF effects. As an example, in Table 7.1 and the later summary tables of Draft 3, "chance", "bias", and "confounding" are treated as independent all-or-none explanations of observed data patterns. This treatment is erroneous for two major reasons: First, each of these explanations (as well as the others in Table 7.1) may easily be partial explanations that together produced the observed data patterns; second, these explanations are not independent. Given that all of the studies at issue were far from perfect, we know that chance, measurement error, selection bias, and confounding each had some influence on the observed data patterns. Each source of error could have affected those patterns along a continuum that ranges from downward to upward bias in the observed EMF-disease association; these error sources would have also distorted apparent dose-response shapes.

A key issue of policy relevance is whether the net (combined) impact of all sources of error on our estimate of EMF effect inflated that estimate from below the action level to what has been observed (assuming that the estimate is above the action level). The probability that the net impact "explains" the observations in this policy-relevant sense is much greater than the probability any one source of error alone explains the observations. This is so, even if the sources of error are independent; and the difference between the two probabilities can grow much larger when the sources are dependent, as they are here. This dependence was shown earlier for selection bias and confounding, and it also holds for all other pairings of error sources (e.g., see Greenland and Robins, 1985, and Greenland, 2000). The consequence is that considering the impact of error sources one-at-a-time in an all-or-none fashion, as done in Draft 3, severely underestimates the probability that the observed summary is explained by errors.

A general derivation of the preceding result is rather technical, but some sense of the proof can be obtained from the following simple example. As of this writing, summary relative risks for measured magnetic fields above 3 or 4 mG versus below 1 mG are around 1.7 with 95% confidence limits of 1.2 and 2.3 (Greenland et al., 2000). Suppose the action region is a causal relative risk above 1.1. We want to evaluate the probability that the net error inflated the summary by a factor of 1.7/1.1 = 1.55 or 55%. Suppose we are sure that confounding alone could not have produced this much or more inflation, nor could selection bias alone, nor could misclassification alone; chance alone could have done so with only 2% probability. If we considered each error source one-at-a-time we then might be tempted to say that alternative explanations could completely account for the observed excess (over 1.1) with only 2% probability (and that 2% arises only from allowing for chance). This impression would be entirely wrong, however, because to evaluate the probability of this complete accounting we must add up the joint probabilities for every possible combination of errors that produce a net inflation of 55% or more.

As an example, we would have to add in the probability that (say) confounding produced 30% inflation and selection bias produced another 30% inflation (both figures are quite possible in light of the results in Hatch et al. (2000) and other data), misclassification produced 30% deflation, and chance produced 25% inflation (very possible, given the confidence interval), to yield a net summary inflation of 1.3(1.3)(1/1.3)1.2 = 1.56, or 56%. We would have to integrate over all such inflationary possibilities (those with 55% or more inflation) that yield the observed pattern of results. Many of these possibilities would have non-negligible probability; the total probability of such inflation could turn out to be considerable under reasonable priors, even if there were zero probability that any one error could come close to producing 55% inflation by itself.

The situation is actually worse than just illustrated because of the dependence of errors, and the fact that various errors can act “synergistically” in a manner such that the above product calculation understates the net bias (e.g., see Greenland and Robins, 1985). For example, “chance” errors can lead to non-negligible upward bias when pivotal cell counts get small, even in an apparently large study, and can especially inflate adjusted estimates as a result (Greenland, 2000).

To summarize: The statement and treatment of questions outlined in Chapter 7, which conceptualizes errors (including biases) as independent all-or-none alternative explanations, leads to severe underestimation of the probability that the net error is enough to have inflated the EMF-disease associations to those observed, given that the true effect is negligible for policy purposes.

The above criticism is not addressed by the considerations offered in Section 7.2 of Draft 3. To avoid "the pitfall of simply adding yes and no answers," the authors provide a continuum for their subjective "likelihood of the pattern of evidence" (p.67, lines 1-5), but offer no continuum for the effects or the sources of error that produced the "evidence" (data) or their interactions. In fact, the process described in Section 7.2 makes no attempt to quantify sources of error and their interactions. Instead, in lines 12-16 of p.67, the authors state that the size of their relative likelihood for a pattern of evidence "depended on how good that stream of evidence was in detecting a cause, if it usually detected a harmful agent (sensitivity), and how good that stream of evidence was in not falsely implicating an agent (specificity)." Thus, they evaluated each "stream of evidence" as if it were a separate effect-detection device with a dichotomous (yes/no) output.

The only point at which the authors seem to become dimly aware of some of the problems discussed above is in lines 31-40 of p.67, where they state that "of course, the answers to these questions [Table 7.1] cannot be mechanically considered in isolation." Unfortunately, they then respond to their own warning by stating that "certain combinations of answers influence our degree of confidence more than the isolated answers would predict," and then go on to confirm that the combinations they consider remain "pro and con answers to the structured questions," i.e., combinations of dichotomies, with no regard to the underlying multidimensional continuum of observational errors and their interactions.

p. 67, lines 44-91:

The authors state, incorrectly, that "the question about chance does not deal with relative likelihood." The question in Table 7.1 is "How likely is it that the meta-analytic or pooled association from these studies is due to chance?" Presumably the authors mean "chance alone" (otherwise their discussion makes no sense).

Unfortunately, the authors then go on to make the common mistake of equating this first question to the question "how probable is the observed, or more extreme, pattern of evidence under the null hypothesis." The first question (from Table 7.1), however, is about the observed "evidence" (data pattern) only, not more extreme patterns; the correct answer to the question is precisely the likelihood for the data! (Royall, 1997). The authors make the further mistake of offering the conventional P-value as the answer to the second question; unfortunately, that P-value assumes no source of error is present other than chance, which is certainly false here. Given their stated goals, the authors should have integrated the statistical likelihood function (not the P-value) into their evaluation of the causal hypothesis.

Section 7.3: General

In addition to the question of "chance" discussed above, Section 7.3 discusses ten highly interdependent questions or considerations. The use of such highly interdependent considerations give the misleading impression that more independent information sources and lines of reasoning have been used than is actually the case. (In technical terms, there are many fewer degrees of freedom of information here than ten.) In several cases the dependency is complete, and as a consequence Draft 3 repeatedly presents the same data evidence in different guises, giving the impression that there is more evidence than actually present.

As an example, the consistency analysis of Draft 3 is nothing more than a transformation of the same data evidence used by the strength and homogeneity analyses. The data basis of the strength analysis includes a summary estimate, call it L_S; the data basis of the homogeneity analysis includes the deviations $D_k = L_k - L_S$ of the study-specific estimates L_k from the summary L_S; and the data basis of the consistency analysis comprises only the study-specific estimates L_k. Because each study-specific estimate L_k equals the deviation D_k plus the summary L_S (i.e., $L_k = D_k + L_S$), the consistency analysis introduces absolutely no new data evidence; hence, by including consistency as an independent consideration, Draft 3 "double counts" the data evidence provided by the study-specific estimates. For example, "consistency" will follow as a consequence of a homogeneous positive association of even modest strength (as in the childhood leukemia data).

The consistency measure used here (simply tallying the proportion of positive results) is highly inefficient, and potentially misleading, for at least two reasons. First, it ignores dose-response: Even if most or all study-specific estimates were positive, we should consider them inconsistent if their trend when plotted against dose was clearly downward. Second, it ignores precision: It would classify results as consistently positive just because several small, highly unstable estimates fell above 1 and a few stable estimates (containing most of the statistical information) fell below 1.

With regard to the remaining questions, it has long been known that considerations of "biological plausibility", "analogy", "coherence", and "dose-response" are all based on the same core of prior (external) information, a core that may be described as biologic or pathophysiologic theory for the effect under study; see p. 24-27 of Rothman and Greenland (1998) and the references therein. That core is highly speculative in the present setting. The same speculative core is used to argue that nonspecificity of the EMF association supports causality (p. 69, lines 33-55). The same core used in the nonspecificity argument is invoked yet again under "evidence from other diseases" (p. 69, lines 56-59). Experimental evidence also affects this core, and conversely the authors of Draft 3 use their beliefs about this core pathophysiology to dismiss negative experimental results (p. 68, lines 51-56). Therefore, I regard the seven considerations from p. 68 line 24 to p. 70 line 34 as representing only a fraction of the information that Draft 3 makes it seem. As described next, there are also specific errors in the treatment of these considerations.

p. 68, lines 53-56:

Here the draft states that "Exposing animals to a pure 50 or 60 Hz sinusoidal magnetic fields [sic] is no more adequate that [sic] attempting to study the protective effect of red wine by exposing the animals to pure alcohol." This is an amazingly defensive attack on the perfectly reasonable strategy of attempting to refute possible explanations for an association. It would be extraordinarily useful to discover that the observed red wine association with lowered CHD risk is not explainable as a pure alcohol effect; this discovery would eliminate one causal hypothesis and would provide a rational basis for directing resources toward other explanations, such as other components of red wine and various sources of error in the observed association. Likewise, if experiments

did establish the harmlessness of pure 60 Hz sinusoidal fields, it would eliminate one causal hypothesis and would provide a rational basis for focusing on other field components and on sources of error.

p. 68, lines 53-56:

The argument given here in item 2 involves a self-contradictory application of specificity, and also a purely mathematical error in its characterization of selection bias. The latter error surfaces repeatedly in the summary tables in later chapters, and exaggerates the posterior confidence in an effect by incorrectly dismissing potential sources of selection bias.

The self-contradiction arises because item 2 claims, with no supporting background information, that "it is much less plausible to believe that, among the factors correlated [with] EMF, there could be an unidentified risk factor for each of the many endpoints associated with EMF exposure." This is a shocking example of transforming ignorance into prior information. Little is known about the risk factors for the endpoints here, let alone whether their associations with EMF are artefactual. The authors are thus invoking Hill's specificity condition against every non-EMF factor, known or unknown, to claim that the same criterion should be reversed for EMF only. In other words, the specificity criterion is invoked to refute itself! The only claim of evidence in support of this argument is a statement that just one known leukemogen, benzene, is "not a credible confounder for breast cancer or ALS, which are not correlated [with] benzene." No studies are cited in support of this claim, and I am doubtful that any valid epidemiologic analysis exists that has exonerated benzene as a risk factor for these outcomes or the other endpoints with any confidence. Indeed, given the extreme rarity of ALS, I would question whether there is much data information at all about benzene and ALS.

The concern about whether the diseases share common risk factors is irrelevant to the selection-bias issue; hence Draft 2 is wholly erroneous when it makes claims such as "it is less plausible to believe that selection bias in favor of higher versus lower social class could explain all the associations, because social class is a risk factor for some diseases and a protective factor for others." It has been known for over 50 years that a factor related to exposure can produce selection bias in case-control data (which compose nearly all the data), even if it does not

affect the disease under study. All that is needed to get bias is that the factor affect the selection or participation rates among controls differently than among cases. When this differentiality occurs, control of the factor will be needed to remove the resulting bias. Indeed, this problem is the reason one needs to adjust for case-control matching factors related to exposure, even if those factors do not affect disease risk (Rothman and Greenland, 1998, p. 151). The consequence is that a shared bias across EMF studies with different endpoints could occur easily if there are uncontrolled or poorly controlled factors that are associated with magnetic-field measurements and with selection or participation, even if there are no shared risk factors.

p. 71, line 34:

The Draft here confuses an alpha level with a P-value: the parenthetical "(p=.01)" should be either "(p<.01)" or "(alpha=.01)"; see Rothman and Greenland (1998, p. 186).

p. 71, line 40:

There appears to be an omission of words; perhaps "to have an excess of at least one..." was meant.

Section 8.1, especially p. 73, lines 1-10:

The summary figures, tables and estimates are poorly described, and make no sense biologically because the odds ratios ("RR") are based on a wide variety of exposure measures and reference points:

1. Figure 8.1.2 and Table 8.1.2 mix odds ratios based on measured or calculated fields with odds ratios based on wirecodes; the categories for the latter wirecodes are not even given.
2. The reader is not informed that the "wirecodes" used in the plotted studies are not at all comparable, or that there is severe heterogeneity among studies that use comparable wirecodes (Greenland et al., 2000).
3. Exposure units for Figure 8.1.1 and Tables 8.1.1 and 8.1.4 are not given at all.

4. No definition is given for the "individual odds ratio mean" in the Table headings; what is this supposed to be a mean of? (Note arithmetic means of odds ratios are poor summaries because of the ratio scaling of the odds ratios; some type of geometric mean would be needed.)
5. In Table 8.1.2, what does the "+" on Verkasalo signify? That it is a cohort study?
6. I suspect a mistake has been made in Table 8.1.3 where the "RR" and "CI" for the four studies under "Proximity to Sources" are all identical to the hundredth's place.
7. Why were the results from the Coghill et al. study of magnetic fields and childhood leukemia omitted from Figure 8.1.2 and Table 8.1.2?
8. Why was the mixed-cancer study by Myers et al. included? As far as I can tell, Myers et al. supplied no breakdown of the "non-solid tumors" by exposure and leukemia status; it thus appears that the counts reported here as leukemia cases in Myers et al. are actually a mixed case group.

Figure 8.1.2 and Table 8.1.2 also exclude several wire-code odds ratios whose reports were available to the DHS reviewers, including those from Green, Linet, London, and Savitz; while the subjects contributing to these results largely overlap with the subjects contributing to the measured-field odds ratios from those studies, their inclusion here would allow the reader to see how well the two measures agree within the same study. As it happens, that agreement is poor (Greenland et al. 2000), which further calls into question the propriety of combining measured-field and wire-code odds ratios in the same table or figure.

The extent of ambiguity and noncomparability in the presentation makes it difficult to evaluate the internal coherence of the results, especially for adult leukemia. We are told later (on page 94, Table 8.2.6) that "Kheifets (2001) found that adult occupational studies were not heterogeneous," but that Kheifets citation is on brain cancer, not leukemia; perhaps Kheifets et al. (1997) was meant. Without any exposure levels to attach to the adult studies, no meaningful summary or evaluation of their coherence is possible; for all a reader can tell, studies with highest exposure levels may have yielded the lowest relative-risk estimates.

Equally worrisome is that (unlike the childhood leukemia studies) the adult studies encompassed a broad range of designs, including cohort, case-control, SIR, PIR, PMR and MOR studies; these designs have potentially very

different validity properties (Rothman and Greenland, 1998, Ch. 5-7), yet there is no discussion of whether dose-adjusted results varied with the designs, or with other relevant study properties (such as measurement type or vulnerability to bias). Given that the Draft 3 authors earlier had used a "complex mixture" argument to dismiss negative experimental evidence, it is especially curious that they made no attempt here to see if the occupational study results varied with the exposure characteristics or measures.

p. 89, A7 and F3:

"Canadian studies of childhood leukemia are heterogeneous from other studies." This statement is wrong. As discussed in Greenland et al. (2000), the apparent difference of the McBride et al. study from non-Canadian studies was only due to categorization differences, the main portion of the Green et al. study data was not reported in enough detail to determine whether it was truly different from others, and the personal-monitor substudy of Green et al. is so small that any difference is easily ascribed to chance.

p. 89, F1:

"No bias candidate is common to all studies." This is a purely subjective claim by the authors, not a matter of fact, and is disputed by some skeptics. But the claim is not very relevant: Even if some biases have inconsistent direction, the primary concern is whether the net errors in the data summaries are positive. There is in fact accumulating evidence (as yet unpublished) that participation bias may have inflated estimates from at least some of the non-U.S. studies, as well as evidence of such bias in some major U.S. studies (e.g., Hatch et al., 2000). The existing uncertainty about bias should reduce our posterior certainty (see Appendix).

p. 89, F2:

It is claimed that the opposite asymmetry of the Savitz and London control-specular matrices proves "that control-selection biases were in opposite directions." This claim of proof is naive: First, as noted by Zaffanella et al., the differences were well within chance expectations; second, also as noted by Zaffanella et al., it is possible

that local factors could have affected the control specular distributions differently in the two studies, especially given the large differences in power-line distributions and housing patterns in Denver and Los Angeles.

p.89, C2:

As shown in the Appendix, although it is true that nondifferential misclassification would deflate the point estimate, properly allowing for the unknown misclassification in these studies would also be likely to drive down the lower limit of the 95% posterior interval.

p. 89, F7:

The Draft claims that "There are no arguments in favor of consistent upward bias." That is false: For the childhood-leukemia studies, many researchers in this area would still allow for the possibility of general upward participation bias, and have supportive data. The sampling of citations given here (on p. 90 of Draft 3) are biased: Several relevant studies have found substantial associations of SES, housing factors, or participation with magnetic fields or wire codes, including Hatch et al. (2000), Bracken et al. (1998), Spinelli et al. (2001), and as yet unpublished work in the U.S., Canada, and Europe. As explained earlier in my criticism of p. 68 of Draft 3, the claim that SES is "virtually not associated with childhood leukemia" is irrelevant here, because SES-related nonparticipation can produce arbitrarily large bias even if SES is not a risk factor.

The Delpizzo (1997) reference, cited to claim that SES and "exposure" are not associated, was not in the reference list of my copy of Draft 3; however, its relevance is questionable because what matters is not whether the association exists in California, but whether it existed in the source populations in which the studies were conducted.

Finally, in what appears to be a very bizarre analysis, the authors seem to indicate that they used residential exposures of childhood brain-cancer cases as a control (reference) for occupational exposures of adult leukemia cases (London, 1994). Perhaps they meant London (1991)?

p. 91, F1:

The claim is made that "All known, suspected, and even speculated confounders were controlled for in every study since Wertheimer and Leeper." This claim is utterly false. Few studies even measured (let alone controlled) most of the "known, suspected, or speculated confounders" (e.g., few or none controlled traffic-density or air-pollution measures), and most of the measured covariates were either measured or controlled in such a crude fashion that the adjustments made would not have removed most of the confounding by the variable (it has been known for over 20 years that mismeasurement of confounders can ruin the ability to adjust for them and can produce spurious associations; see Rothman and Greenland, 1998, p. 133).

p. 91, F3:

The cited Pearson et al. study provides only extremely weak data on the matter.

p. 91, F4:

This item overlooks that, in the studies by headed Green, Linet, McBride, and Savitz, the crude SES measures are associated with childhood leukemia (low SES at highest risk, sometimes with odds ratios of 3 or 4). These extensive case-control data directly contradict the Draft 3 claim that "SES is virtually not associated with childhood leukemia" (p. 90), a claim that cites as support only Reynolds et al. (2001), a tiny (90 cases) case-control study that had only ecologic (neighborhood) SES measures, not individual data. This claim is a striking example of the extremely biased literature citations given in the summary evidence tables in Draft 3 (other examples are given below, e.g., regarding p. 95). Even if these SES-leukemia associations turn out to be due to response bias, as suggested by Spinelli et al. (2001), that finding would demonstrate a potential for response bias in the studies large enough to explain the magnetic-field-leukemia associations.

p. 91, F5:

"There are convincing quantitative argument [sic] against the plausibility of confounding by an unknown factor (Langholz, 2001)." This statement is a good example of the one-at-a-time, all-or-nothing approach that drives the conclusions of Draft 3. The Langholz citation analyzes only the question of whether a single confounder alone could explain the VHCC odds ratio of 1.9 from the Los Angeles study. That analysis ignores every other source of error, including chance, and so assumes away error combinations and interactions. A Bayesian analysis of the same problem (see Appendix) reveals that consideration of unknown confounding should reduce posterior certainty.

p. 91, C1:

"The existence of a strong confounder...in every population studied is less plausible than accepting EMF as a causal factor." This comment is a purely subjective a priori preference of the Draft 3 authors. It is also not very relevant to a sound risk analysis, because the real question is whether, on the balance, confounding from multiple sources even partially contributed to the observed patterns.

p. 91, C2:

The second sentence here is yet another a priori purely subjective opinion of the authors, and is even more irrelevant than that in C1. Few would doubt that at least some net confounding (however slight) is present in the summaries, and then the only question is whether that net confounding is upward or downward. Given how little is known (mostly only that some residual upward confounding by some housing-related factor seems plausible in light of the data cited above), one could more plausibly argue that, at present, net upward confounding seems somewhat more likely than net downward confounding, though the amount of that confounding seems unlikely to be large. Even so, allowance for the possibility would reduce posterior certainty (see Appendix).

p. 92, F1:

This comment concerns only the point estimate and as such is misleading, for reasons detailed later (see next comment and the Appendix).

p. 92, C1:

Based only on the expected effect of nondifferential misclassification on point estimates, it is claimed that low relative-risk estimates should not decrease confidence in the causal hypothesis appreciably. Taken literally, this is a prescription to discount negative evidence, because it leaves no situation in which observations could "decrease confidence appreciably" for the causal hypothesis (all estimates are either high or low).

A more subtle but serious fallacy embodied in C1 and elsewhere in Draft 3 is that the authors are incognizant of the impact of unknown misclassification on the posterior spread of the relative risk (as measured, say, by the posterior variance of the log relative risk). As shown in the Appendix, that impact can easily result in reduction of the lower 95% posterior limit, even when it greatly increases the point estimate (posterior center), as one would expect here. Consequently, accounting for unknown misclassification patterns (as in this subject) can reduce posterior confidence in the causal hypothesis, even when it increases our point estimate! This apparently paradoxical result is a good example of why "simple, intuitive" reasoning about bias, as used here, can be so misleading in development of posterior inferences.

p. 93:

For reasons given earlier, I regard the "consistency" analysis used by Draft 3 as a recounting of the same data evidence used for analyzing strength and homogeneity, but in a way that is inefficient and potentially misleading. Hence this analysis should be given no weight at all for forming posterior inferences. At most, it is just a rebuttal to any naive significance-test vote-tallying (as in A1) that unsophisticated skeptics might use.

p. 94, F1:

The Kheifets (2001) citation is on brain cancer, not leukemia. Perhaps Kheifets et al. (1997) was meant; if so, one must question why it is presented in the "For Causality" column: That study analyzed the relation of study results to study characteristics that could influence validity; their abstract concluded that "while most studies present a small elevation in risk, the apparent lack of a clear pattern of exposure to EMF and risk of leukemia substantially detracts from the hypothesis that measured magnetic fields in the work environment are responsible for the observed excess risk," and also that "some evidence of publication bias was noted." The Draft 3 authors need to justify their apparent contradiction of these conclusions.

p. 95, F1:

This paragraph ends with the statement that the Greenland et al. (2000) pooled analysis showed "a weak trend for threshold below" 3 mG. This statement would be reasonable if "for" were replaced by "or"; perhaps "or" was intended.

p. 95, F2:

It is claimed that "The adult studies are consistent with a sigmoid risk function." This statement is highly misleading because the data are consistent with many other shapes as well (including those more consistent with error, such as a rise and fall), and the authors of Draft 3 neither present nor cite any dose-response meta-analysis of adult data. Kheifets et al. (1997) did such an analysis and concluded that "the apparent lack of clear pattern...substantially detracts from the [causal] hypothesis..." The Draft 3 authors attempt to explain away this problem with a saturation (sigmoidal) hypothesis. Because there are no biologic data to show that saturation should occur at such low exposure levels, this hypothesis strikes me as a degenerative (in the sense of Lakatos) post-hoc attempt to explain away a data feature not supportive of causality.

Under F2, the authors go on to claim that "clearer associations are found with the highest exposure group in each study's population." That claim is patently false, as Figure 4 of Kheifets et al. (1997) shows; as Kheifets et al. note, only a minority of the studies in the figure show the highest relative risk in the highest category.

As F2 continues on p. 96 of Draft 3, the authors cite only one positive study each for work-and-home and duration measurements. It appears from Kheifets et al. (1997), however, that other studies had duration information; what were their findings?

p. 95, A3 and C3:

The second and third sentences of A3 are simply incorrect, because the data are in fact consistent with a quadratic dose-response (just as they are consistent with many other curves).

p. 97, Table 8.2.8, C3:

As noted earlier, the Green et al. study is not at all puzzling because the reported data are statistically compatible with almost anything.

p. 99, Table 8.2.15:

This table is the most misleading of all for the reasons discussed earlier, especially regarding Chapter 7:

1. "Chance not a likely explanation": "Increases confidence". First, the authors considered only P-values, not data likelihoods, which (as explained by Royall, 1997) exaggerates the evidence against the null. Second, chance was considered in isolation, disregarding its potential interactions with other sources of error.
2. "Bias not proven": "Pulls down confidence slightly, if at all". The authors did absolutely no sensitivity (or other) analysis that would allow them to dismiss the impact of unknown biases on posterior inferences. In fact, its impact could be considerable even if "not proven", and its possibility should reduce posterior certainty (see Appendix).
3. "Confounding not identified": "No impact". This conclusion is just fallacious. As shown in the Appendix, accounting for unknown confounders must decrease posterior certainty.

4. "Consistency": "Increases confidence quite a lot". As shown earlier, this is simply a repeat of the homogeneity and strength evidence; entering it here amounts to double-counting the main positive stream of evidence.
5. "Experimental evidence": "No effect or increases confidence somewhat". That this mostly negative body of studies should increase confidence at all is never demonstrated, and in fact most other EMF researchers I have talked to regard it as decreasing confidence.
6. "Specificity (associated with other subspecies of this endpoint or other endpoints)": "No impact or slight increase in confidence". First, the parenthetical phrase and the text argument describe nonspecificity, not specificity. Second, as discussed above, the argument used to claim non-specificity is "more possible" under causality than under noncausality invokes specificity against all alternatives to causality whether known or unknown. Such a twisted argument should only decrease our confidence that the authors of Draft 3 were impartial in their evaluation. Given that specificity has been rejected as a consideration by many authors over the last 40 years (e.g., Sartwell, 1960, and Rothman and Greenland, 1998, p. 25) and that it is somewhat redundant with plausibility considerations, it is not even needed here.

p. 100, lines 8-21:

"The net effect of these unidentified biases [and confounding] should be small." This statement is an incorrect confusion of net expectation with expected deviation (e.g., expected absolute or squared deviation from truth). There is no data basis for expecting miraculous complete cancellation of error sources, and proper allowance for incomplete cancellation will decrease posterior confidence. The deep fallacy in the thinking displayed here can be seen in a simple example. Suppose our posterior distribution for net systematic error is normal (Gaussian) with mean zero and nonzero standard deviation S_{bias}. This implies that we have no good idea of the direction of net bias (including confounding), but we are not so foolish as to believe that all such errors have cancelled perfectly. Then the amount of squared systematic error we should expect is S_{bias}^2 , the variance of our systematic-error distribution. Assuming that systematic and chance errors are independent (which is not realistic, but that is what is assumed by the Draft 3 authors), we must add S_{bias}^2 along with sampling error into our

posterior variance calculations to get a valid posterior "confidence" interval. None of this argument requires that there be "a bias common to all or most studies," nor does it require a bias "that can explain away the association," as Reviewer 1 seems to mistakenly think (lines 8-9).

p. 100, lines 21-29:

As explained before, "consistency" as defined in Draft 3 is just a combination of strength and homogeneity: A homogeneous association logically must display some consistency if it is even weakly positive; "consistency" can also arise from heterogeneous associations, if on average the associations are positive; hence "consistency" is logically weaker than strength and homogeneity. Thus, what Reviewer 1 claims is "the strongest factor arguing for causality" is nothing more than a weaker version of two other considerations not viewed as strongly by Reviewer 1! This fact shows that the reasoning used by Reviewer 1 is not only statistically fallacious, but is logically fallacious as well.

p. 100, lines 49-51:

Regarding experimental evidence, Reviewer 1 claims that "false negatives are a virtual certainty." False negatives (failure to detect a true effect) can be a certainty only if a true effect exists, the very proposition under debate. Also, false negatives can be "more probable" than false positives (lines 51-52) only under some (not all) prior probabilities for the sizes of the effects. These comments suggest to me that Reviewer 1 has an even stronger prior in favor of effects than he described.

p. 100, 63-64:

Whether trends were statistically significant or not within single studies is irrelevant for a meta-analysis, pooled analysis, or risk assessment. What is relevant here is whether the study-specific trends are reasonably homogeneous with one another, and whether the pooled trend is reasonably monotone. Both conditions are satisfied for childhood leukemia, and in my opinion this fact is the aspect of the childhood-leukemia data most

supportive of causality. For the adult-leukemia data the published information appears too crude to provide as much support for causality based on these considerations.

p. 101, lines 20-22 and 28-29:

I regard the extremely high posterior confidence of Reviewer 1 as largely a product of the systematic evaluation errors and fallacies documented above. Quite possibly, his biased evaluation was abetted by a prior that was not successfully decontaminated of familiarity with positive results. The 99% posterior confidence he offers for EMF causing childhood leukemia is especially troubling, given all the unresolved issues of selective participation that remain.

p. 101, lines 54-55:

Reviewer 1 states that biases "are random events that are accounted for by an appropriate statistic [sic] test, such as determining the p-value using a sign test." This is one of the most ridiculous erroneous statements I have ever seen in my 25 years experience as a methodologic and statistical reviewer and editor. If it were true, observational researchers would never have to worry about biases, knowing that the statistics took them into account. The statement is, however, quite false, and confirms that the author has no sound understanding of epidemiologic analysis.

First, as suggested by the use of "systematic error" as a synonym for "bias" (including confounding), biases are not "random" in the repeated-sampling sense assumed by conventional statistics such as P-values and confidence intervals. In fact, the key problem in epidemiology is that such systematic errors produce bias in ordinary statistics, <u>whether those errors are known or not</u>, even if we have "no reason to believe that biases in one direction are more likely than those in the other." One basic illustration of why this is so is given by Greenland (1990), but every competent epidemiologist knows that conventional statistical methods (like sign tests) account only for chance errors, nothing more. Accounting for systematic errors requires building a

plausible model for the error (bias) sources, and at a minimum conducting a sensitivity analysis, which was done nowhere in Draft 3.

p. 101, lines 59-60:

The hypothesis tested by the sign test (and any conventional statistical test) is not that "EMF exposure conveys a risk greater than 1;" rather, it tests the noncausal null hypothesis that there is no association (causal or noncausal) of EMF exposure with leukemia in any of the source populations of the studies. The test takes absolutely no account of systematic errors, so that "p<0.001" only says that chance alone would only rarely produce such a pattern of associations, something we already know. But it in no way rules out chance as a possibly major contributor to the pattern (along with other sources of association, including EMF effects and systematic errors).

p. 101, lines 65-66:

Here at last Reviewer 1 concedes the possibility that systematic errors may partially explain the observations. Next he should consider the possibility (not certainty by any means, but one reasonable possibility nonetheless) that some combination of systematic errors and chance may have produced most or even all of the associations.

p. 102, lines 1-13:

Here, Reviewer 1 attempts to rationalize his all-or-none treatment of potential bias sources. It contains several errors. First, the childhood leukemia results have not emerged from a variety of study designs: All the studies with informative data have been population-based case-control studies, and so are vulnerable to the participation and response biases that are of ongoing concern to competent researchers in this area. Second, to explain the observations, it is not at all necessary that systematic errors "explain enough positive studies so that the remaining ones can be attributed to chance"; many other patterns of systematic error and chance would suffice, such as a continuum of mixtures of the two. Third, it is just naive to claim, as does Reviewer 1, that powerful and

consistent sources of error could not "remain unidentified over 20 years of efforts": For example, the powerful and consistent SES-leukemia association across these studies was not systematically examined until fairly recently, and was not even noticed (let alone discussed) by the authors of Draft 3 (who are presented as experts on the topic).

p. 102, lines 25-36:

This passage repeats or assumes several erroneous interpretations noted earlier. Prominent is the erroneous claim that the occupational studies "are slightly more homogeneous than those of childhood leukemia, sharing a somewhat more similar environment..." In reality, heterogeneity of the work environments in these studies is profound (some studied welders, some studied painters, some studied very mixed groups, and so on), and many potentially confounding exposures were present and poorly measured, or unmeasured in most of these studies. Compared with the childhood-leukemia literature, a higher proportion of occupational studies show no association or a non-monotone dose-response, and publication bias remains a possibility (Kheifets et al., 1997). None of these issues are even mentioned by Reviewer 1.

Reviewer 1, General:

In light of the severe mistakes of statistics and epidemiologic reasoning, the logical fallacies, and the gaps in literature knowledge displayed by Reviewer 1, as documented above, his evaluation should be discarded as untrustworthy and unreliable.

p. 102-103, Reviewer 2, General:

Reviewer 2 does not make as many or as severe mistakes as Reviewer 1, and is much more scientific and restrained than Reviewer 1. Like Reviewer 1, however, he fails to properly account for possible multiple sources of error and their interactions, taking a one-at-a-time approach, and does not address the data connecting SES-related factors to childhood leukemia and to participation bias. If examined as thoroughly as they deserve, the

preceding considerations should reduce the confidence of Reviewer 1: Because Reviewer 1 gave on a 55% (15%-80%) posterior probability for EMF causing childhood leukemia, his revised posterior might be below 50%.

As for adult leukemia, I would suggest that Reviewer 2 may have over-rated the general quality and consistency of the studies. "State of the art" (p. 103, line 30) in these settings seems to mean only "the best that can be done at this time." Based on my reading of secondary sources such as Kheifets (not "Kheifetz"), my impression is that nearly all of these studies involve at best very crude measurements of exposure, potential confounders, and potential selection factors, and that (unlike for childhood leukemia) differential errors and publication bias are not minor possibilities. Furthermore, the between- and within-study consistency with causality does not appear to be as great as for childhood leukemia (e.g., see Kheifets et al. 1997). Thus it is hard for me to see why Reviewer 1 gives a posterior probability for EMF causing adult leukemia that is only slightly below that given to childhood leukemia (52% for adult versus 55% for childhood).

p. 103-104, Reviewer 3, General:

Reviewer 3 does not provide as detailed a discussion of her reasoning as Reviewer 1 and 2. It is however clear that her evaluation shares several mistakes with the other reviewers. In particular, like Reviewer 1, Reviewer 3 gives a very facile dismissal of biases (p. 103, lines 57-59 and p. 104, lines 3-5), showing no awareness of the unresolved concerns and data on SES and participation bias in the childhood leukemia studies.

Reviewer 3 also incorrectly states that "the probability of chance contributing to the positive effect is low" (p. 104, lines 2-3). In reality, all that has been shown by the statistics is that chance alone is not likely to have produced the observed association. Chance almost certainly has contributed one direction or the other to its size; the probability that this contribution is upward is 50%, a fact that should not be ignored given the potentially large amount of random error that may be present (e.g., the 95% confidence interval for the Greenland et al. (2000) summary odds ratio ranges all the way from 1.2 to 2.3, a nearly two-fold range). The oversights documented above, if properly accounted for, should greatly reduce the posterior probability of Reviewer 3 for the EMF effect on childhood leukemia, to something more compatible with the evaluation of Reviewer 2.

p. 105, Table 8.4.2:

The table claims that "if we group Greenland's data into 4 exposure classifications, thus narrowing the confidence intervals, we virtually rule out the no-threshold function (see Dose Response chapter)." As shown below in my criticisms of the Dose Response chapter, this claim is based on an erroneous statistical analysis of the confidence intervals. In reality, the available data are quite incapable of ruling out a no-threshold function, and the "evidence of a 2-3 mG threshold" is so statistically unstable as to be useless. The most that can be said is that there is no evidence of elevation of risk below 2 mG, and that if a threshold exists (a big "if") it could easily be at 3 mG or even higher.

Contrary to the claim made in the table, neither the regression analyses nor the grouped data should be construed as suggesting that a plateau exists: Above 5 mG there are so few subjects that almost any shape of the response curve is highly compatible with the data.

p. 106, Table 8.4.5:

It is true that "little is known about the risk factors" for leukemia, but it is wrong to claim that "the proposed risk factors are not strong and do not account for much of the incidence," because most of those factors have received much less study than EMF and hence there is little evidence on which to base that claim. In reality, the very same North American data that contribute the bulk of the EMF-childhood leukemia evidence show an even stronger association of childhood leukemia with certain SES factors than with EMF; these observations suggest that either some risk factor much stronger than EMF exists, or else very strong selection bias is at work in these studies.

p. 108, Table 8.4.9:

Regarding "Italy": Statistical power of a single study is irrelevant for meta-analytic purposes.

Regarding “Japan” and “Germany”: It has been reported in unpublished presentations that both of these studies are experiencing severe problems from participation refusal among controls.

Regarding “Washington”: The study described sounds worthless given the rarity of relevant exposure. Note that “Principal” is misspelled.

p. 109, Section 8.5.1:

Despite the erroneous data interpretations in Tables 8.4.3 and 8.4.5, the conclusion given here is reasonable.

p. 109, Section 8.5.2:

Given that some proposed (and expensive) remediation would be futile if contact currents are responsible, I would say “should explore” rather than “could explore” in the first line of this section.

Chapters 9-18:

I was not requested to review these chapters. Nonetheless, these chapters would suffer from the same statistical and logical fallacies in the general methodology and reasoning process that afflicted Chapter 8; see especially my general critique and my criticisms of the general Draft 3 methodology in Chapter 7. Most worrisome and pervasive are the double-counting of data via the use of the consistency criterion, the all-or-none treatment of errors, and the failure to understand the impacts of unmeasured error sources on posterior probabilities (see Appendix).

As an important example, in Chapter 9 the reviewers commit most of the errors and fallacies in formulating their confidence about the effect of EMF on adult brain cancer risk that they committed in Chapter 8. These errors uniformly drive upward their confidence. Most striking is the logical fallacy in Table 9.2.15, in which both

"Strength of association" and "Homogeneity" are evaluated as having "No impact to slight decrease" on posterior confidence, yet their deductive consequence, "Consistency", is said to have "increased" the confidence. The authors fail to grasp that having even a small preponderance of upward bias can lead to satisfaction of their "consistency" criterion because the latter only depends on the very unstable discrete property of whether an estimate is above or below 1; the parent criteria of "strength" and "homogeneity" are more robust to such problems because they do not discretize the evidence.

In Chapter 9 as in Chapter 8, the upward bias in posterior confidence is extreme for Reviewer 1, producing a "median" 98% (70% to 100%) confidence, a ridiculously high assessment in light of the data evidence. Reviewer 2 is again much more reasonable and cautious, giving a 52% (30% to 70%) confidence; this confidence would be pulled downward somewhat by consideration of the above problems, probably to below 50% (since it is not much above that already). Reviewer 3 is again like Reviewer 1 in displaying severe overconfidence, but is not as extreme.

Chapter 20, General:

This chapter presents statistically erroneous analysis of dose-response and attributable fraction for magnetic fields and childhood leukemia, which forms the basis of some incorrect claims cited earlier. The errors in the analysis occur across a sequence of calculations, and so compound one another. All act to falsely increase the apparent precision of the results and distort the apparent dose-response. These errors are described below in the order they occur in the chapter, not the calculation:

p. 298, lines 14-15:

The use of category midpoints is biased when, as with these data, the exposure distribution is extremely skewed (e.g., see Greenland, 1995 for a discussion of the relation of category coding to apparent dose-response). In this case, the bias is toward exaggerating the appearance of a threshold. The unbiased center points under a linear response are the control category means, which are certainly lower than the midpoints for categories 1-2 mG and

2-4 mG. As one can see from Figure 10.1.1, if we move down the estimates for the closed-ended categories without moving the top estimate, the apparent trend will bend less sharply in category 3.

As for the top category, at line 15 the authors say that the "midpoint" was set at 5 mG, but the Figure shows it plotted at 6 mG. The latter value is almost certainly a better choice, given that the mean exposure reported by Greenland et a. (2000) above 3 mG was 5.8 mG and that moving the lower boundary up to 4 mG could only increase the category mean (probably to above 6 mG, in which case a plot of trend against means would bend even less sharply in category 3).

p. 298, lines 16-17:

"Figure 1 plots the fraction of study subjects in these exposure categories who are cases": This is a biased way to plot case-control data. As explained, for example, by Greenland et al. (1999) and illustrated in Rothman and Greenland (1998, Ch. 17), one must plot case-control ratios, not case fractions, to obtain undistorted trend shapes. The reason for this need is explained, for example, in Chapter 7 of Rothman and Greenland (1998): The controls, not the totality of study subjects, represent the source-population exposure distribution to which the case-control estimates are supposed to refer.

p. 298, lines 17-18:

"The number of controls per case was standardized for all studies." It is not explained how this "standardization" was done. In any event, standardization of control numbers rather than standardization or modeling of the case-control ratios or odds ratios (as should have been done instead; see Rothman and Greenland, 1998, p. 264-265, and Greenland et al., 1999) will bias the point estimates in an unpredictable manner, and will also downwardly bias (deflate) the standard-error estimates for the plot because it takes no account of between-study variation. This deflation implies that the intervals in Figure 10.1.1 are too narrow to an unknown degree.

p. 298, lines 24-33:

This argument for rejecting linearity is completely incorrect. First, the plot is distorted away from linearity by the use of midpoints. Second, the intervals are too narrow, as just explained. Third, as explained by Hastie and Tibshirani (1990, sec. 3.8.2) and Greenland et al. (1999), one cannot test the fit of a line or any other curve by seeing whether or where it passes within the plotted confidence intervals. This last mistake can lead to either incorrect acceptance or rejection of a curve, and occurs because the plotted intervals are only pointwise, not global, confidence sets. For an explanation of the distinction between pointwise (single) and global (joint) confidence regions, see Rothman and Greenland, 1998, p. 323-328.

The problems just described and the standardization problem described above could have been avoided easily had the reviewers simply entered the study-exposure specific case counts and control counts in a logistic regression program. One could have then modeled the dependence of case status on study (as a set of indicator variables) and on indicators for field categories, then repeated the analysis replacing the field-category indicators by various functions of estimated control means in the categories. This process would have provided valid confidence intervals for plotting trends against category means, and would have provided model loglikelihoods to allow testing curve fits. Had they done a correct analysis such as that just described, they would have discovered (as did Greenland et al., 2000) that the data are statistically compatible with a line, though not as much so as more downward trends.

p. 299, lines 32-50:

The population attributable-fraction ("PAR%") estimate given here is upwardly biased because of the dose-response estimation errors described above. The calculation of the 95% confidence interval in lines 48-50 is invalidly narrow for several reasons, one being that it does not properly account for model uncertainty (use only of lines that "fit within the [plotted] 95% CI" is completely inadequate), another being that it takes no account of uncertainty in the exposure distribution. The model uncertainty issue is especially important, because the estimate is extremely sensitive to whether the curve allows effects below 2 mG. For correct estimation and

confidence-interval methods see Rothman and Greenland (1998, p. 295-297), who give categorical methods, and Greenland (2001a), who gives modeling methods.

TECHNICAL APPENDICES

1. Accounting for Sources of Uncertainty: General

Many of the specific criticisms given in my critique can be viewed as corollaries (specific to the present subject) of the following basic decomposition theorem, as applied to Bayesian analysis and risk assessment:

Theorem: For any two random variables X, U with the required expectations,

$$E(X) = E_U[E(X|U)], \text{ and}$$

$$Var(X) = E_U[Var(X|U)] + Var_U[E(X|U)].$$

In the present subject, X represents the vector of target effects (the effects of magnetic fields on the diseases of interest) and U represents a vector of unknown parameters that affect our expectation for X, such as the parameters that determine confounding, measurement error, and selection bias.

Because the second variance component $Var_U[E(X|U)]$ will be positive, use of the first variance component $E_U[Var(X|U)]$ alone to measure uncertainty about X will understate that uncertainty. If U contains parameters capable of strongly affecting our expectation for X within plausible ranges for those parameters, as when X is an EMF effect, $Var_U[E(X|U)]$ will be large and hence the understatement of uncertainty from neglecting this variance component will be large. Sensitivity analyses can detect this problem, although only for recognized components of U. Unfortunately, Draft 3 fails to recognize the existence of the problem and provides no sensitivity analyses that would hint at its magnitude.

Draft 3 fails to properly account for uncertainty about unknown bias parameters U in both the prior and posterior assessments. Instead of estimating both uncertainty components, the reviewers arrive at an assessment u of U and then offer uncertainty assessments based on X given U=u. This practice results in two downward biases in the assessments: First, it replaces the first uncertainty component $E_U[Var(X|U)]$ with Var(X|U=u); the assessed values of U used in Draft 3 lead to underestimation of the first component. Second, as mentioned above it neglects entirely the second uncertainty component $Var_U[E(X|U)]$. Furthermore, several relevant parameters are overlooked (effectively set to zero), partly through incorrect independence assumptions, which leads to further understatement of Var(X). These three sources of downward bias in assessing Var(X) lead to the severe

overstatement of certainty in the Draft 3 conclusions. In addition, bias in the estimated location of X is introduced by use of E(X|U=u) in place of E(X), which is compounded by the underspecification of U and the regression function E(X|U).

2. Proper Accounting for Unknown Sources of Systematic Error Increases Posterior Uncertainty: A Confounding Example.

On page 99 of Draft 3, Table 8.2.15 states that "confounding [is] not identified," i.e., no one has demonstrated the existence of a confounder that alone would explain the observed association of magnetic fields and childhood leukemia. That statement may be agreeable to most researchers in the field; nonetheless (as discussed earlier), several key North American studies show consistent evidence that some SES-related factor is strongly associated either with risk or selection in those studies. Also, both field strength and SES are related to dwelling type; though the extent of their association with each other is a matter of some disagreement, the observed relations raise a possible avenue for an association of the SES-related risk or selection factor with magnetic fields or wire codes.

The point of these observations is not to argue that uncontrolled confounding or selection bias is present, let alone explains the field-leukemia association. Rather, it is to argue that current data (as opposed to prejudice) do not permit us to rationally exclude all possibility of such systematic error, or even to declare that modest amounts of such error are "improbable", even though we may strongly doubt that the error is large.

In the same line of Table 8.2.15, the authors state that confounding considerations failed to affect their "confidence" in the causal hypothesis. I will here give a formal demonstration that this failure is incorrect under a reasonable Bayesian assessment of uncertainty. This demonstration is merely a special case of the general arguments given in Appendix 1, but may make the points more vivid and relevant. The demonstration will also show how the sort of all-or-none sensitivity analysis given by Langholz (2001) can mislead one into disregarding a relevant source of uncertainty.

Let U be a covariate that is unmeasured, or so poorly measured that adjustment for it is ineffectual, let X be the VHCC wire-code indicator, and let Y be the case indicator for childhood leukemia. Langholz (2001) performs a sensitivity analysis on the following data from the Los Angeles study (London et al., 1991):

	X=1	X=0
Y=1	42	169
Y=0	24	181

The estimate of the crude odds ratio OR_{XY} from this table is 1.87 with 95% limits of 1.09 and 3.23 and an upper one-sided P-value of 0.011; the latter yields a posterior probability for a VHCC effect of 98.9% under a vague prior for the effect, assuming absence of systematic error.

Dichotomizing U as 0,1, we can represent the unobserved stratification of expected counts E_{UXY} in the 3-way table of U, X, and Y by the saturated log-linear model

$$E_{UXY} = c \cdot \exp(a_U U + a_X X + a_Y Y + a_{UX} UX + a_{UY} UY + a_{XY} XY + a_{UXY} UXY)$$

Note that

1. The model makes no assumptions, because it has as many parameters as there are data cells (8).
2. $\exp(a_{UX})$ is the odds ratio relating the covariate to exposure among the controls (or their source population).
3. $\exp(a_{UY})$ is the odds ratio relating the covariate to disease among the unexposed.
4. $\exp(a_{XY})$ is the odds ratio relating the exposure and disease when U=0.
5. $\exp(a_{UXY})$ is the proportion by which the exposure-disease odds ratio changes in moving from U=0 to U=1 ("effect modification" by U).
6. When $a_{UXY}=0$, the XY odds ratio is homogenous across U and $\exp(a_{XY})$ is the U-adjusted odds ratio.

Assuming that $a_{UXY}=0$ (constant XY odds ratio across U), and using the Cornfield et al. (1959) approximation to the crude odds ratio,

$$OR_{XY} \approx \frac{1+P(U=1|X=1)[\exp(a_{UY})-1]}{1+P(U=1|X=0)[\exp(a_{UY})-1]},$$

Langholz showed that UX and UY odds ratios ($\exp(a_{UX})$ and $\exp(a_{UY})$) of at least 5 or 6 would be needed to completely "explain" the observed crude XY odds ratio, in the sense of producing that large a crude association when the adjusted odds ratio $\exp(a_{XY})$ equals 1.

Even if U represents a composite of all uncontrolled factors, such a strong relation of an unmeasured covariate to both exposure and disease seems "improbable." But should the latter improbability of such strong unmeasured confounding leave unchanged or even increase our probability for the X,Y effect relative to what it would have been had we never even considered the possibility of such covariates? NO! Even symmetric allowance for the possibility of bias should reduce our probability that there is an effect.

Why a reduction? Because (assuming homogeneity) the crude XY odds ratio OR_{XY} equals the U-adjusted odds ratio $\exp(a_{XY})$ if and only if $a_{UX}=0$ or $a_{UY}=0$, that is, if and only if either the UX odds ratio $\exp(a_{UX})$ or the UY odds ratio $\exp(a_{UY})$ is 1. Standard confidence intervals for the XY effect implicitly assume homogeneity and that this "either…or" requirement is satisfied. If we replace this extreme assumption about a_{UX} and a_{UY} (which reflects no informed judgement or prior data about unmeasured covariates) with a credible joint distribution, we will see the posterior intervals expand.

For example, suppose we specify $\exp(a_{UX})$ and $\exp(a_{UY})$ as independent lognormals with medians of 1 and 95th percentiles of 5.5, the covariate prevalence in the unexposed noncases $\text{expit}(a_U)$ as uniform, and $\exp(a_{UXY})$ as lognormal with median of 1 and 95th percentile of 1.40. We then obtain approximate posterior 95% limits for $\exp(a_{XY})$ of 1.00, 3.55. This prior specification in no way restricts the size of the adjusted XY odds ratio and assigns only $2(0.05^2) = 0.5\%$ probability to having both the UX and UY odds ratios extreme enough (both above 5.5 or both below 1/5.5) to completely "explain" the crude association. Yet it is sufficient to move 1.00 into the 95% posterior interval for the conditional XY odds ratio, and reduces the posterior probability of an XY effect from 98.9% to 97.5%. This reduction may not seem like much, but that is because of the scale compression from the probabilities being near 100%. On the posterior-odds scale it is a large change: (98.9/1.1)/(97.5/2.5) = 2.3-fold. As we sequentially account for other unknown sources of error, our posterior probability of an effect will be considerably reduced. If we allow for dependencies among the error sources, the reduction can become even greater.

3. Adjustment for Misclassification can Decrease Posterior Certainty Even When it Enlarges the Point Estimate.

When we attempt to correct for likely degrees of field-measurement error using correction formulas (e.g., as in Rothman and Greenland, Ch. 19), we get different results, depending on what we assume for the error rates. How can we allow for the fact that we have only vague ideas about those rates? In Monte-Carlo Risk Analysis (MCRA), also known as Monte-Carlo sensitivity analysis, we

1) repeatedly sample from credible distributions for the validity parameters (error rates),
2) for each parameter sampling, compute a corrected analysis,
3) summarize the distribution of results from the corrected analyses.

When MCRA is done properly, the results can approximate a Bayesian posterior distribution (Greenland, 2001b). As importantly, it can guide us about the impact of accounting for uncertain errors; intuition is based on point estimates, and so can be misleading about interval estimates. Consider the following data on measured fields and wire codes from the four studies used in Greenland et al. (2000) that supplied both measures:

Measured field:	> 3 mG		≤ 3 mG	
Wire code:	VHCC	not VHCC	VHCC	not VHCC
cases	15	40	73	722
controls	8	34	52	910

The table below shows the results from the conventional Mantel-Haenszel analysis of these data, and from one corrected analysis (sensitivity experiment) assuming false-positive and false-negative rates of only $F_p=F_n=2\%$ for classifying subjects as > 3mG; this correction does not account for uncertainty about F_p and F_n. Also shown is the result of an MCRA in which logit(F_p) and logit(F_n) are jointly sampled from a bivariate normal distribution with a correlation of 0.9, means of logit(0.02), and standard deviations of 1 (these make error rates over 10% less than 5% probable, and make the medians of F_p and F_n equal to 2%); also, sampling is independent of disease status but correlated with wire-code status (0.9 correlation):

Error Rates	Leukemia Odds Ratios (95% bounds)	
$F_p=F_n$	field > 3 mG	VHCC
0% (no error)	1.5 (.96,2.2)	1.7 (1.2,2.5)
2% (naive correction)	1.8 (1.1,3.1)	1.7 (1.2,2.4)

MCRA, 2% medians	1.8 (.94,3.5)*	1.7 (1.2,2.4)*

* 50^{th} (25^{th}, 97.5^{th}) percentiles from 6,000 trials

Both corrected analyses show that the order of the field and VHCC odds ratios can be reversed by allowing for even a small amount of field misclassification. But look at the differences in the field-odds ratio interval from the two corrected analyses: The MCRA shows that allowing for our uncertainty about F_p, F_n and their relation to the other variables can expand the interval in a way that more than compensates for the inflation of the point estimate. Thus the naive argument used in Draft 3, that the presence of presumably nondifferential misclassification should increase our certainty that the odds ratio is above 1, breaks down once we allow for realistic uncertainty about the error pattern: The point estimate does increase, as naive intuition suggests, but the standard error increases more rapidly. (The wire-code limits are largely unaffected because wire-code was here assumed to be measured without error.)

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July 27, 2001

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Dear Dr. Collins,

I am responding to your draft report on "... risks from EMF from power lines, and ... appliances" in my capacity as the 1994-96 Study Director for the American Physical Society's study on "Power Line Fields and Public Health." Attached you will find:

- The 1995 APS Statement on "Power Line Fields and Public Health"
- A copy of my paper,"The imprudence of' Prudent Avoidance"'
- First page of the APS ENU background paper www.aps.org/public_affairs/archieves/shtmi, and Amer. J. Physics 64, 974-981 (1996)]

In 1999 1 did a further review of the literature in publishing *Biological Effects oflow-Frequency Electromagnetic Fields* (Amer. Assoc. of Physics Teachers, 1999, 120 pages). The reprints on epidemiology and commentary from the New England Journal of Medicine were very damning of the process in this area.

Your draft report is in opposition to the results of the National Academy of Sciences, the American Physical Society and the American Medical Association. Your tabular results of 50% causality for adult leukemia and 70% causality for adult brain cancer are beyond the pale! I am concerned that a well-meaning process has gone astray. Your efforts will divert public funds to needless mitigation for ELF when you could have saved lives had your efforts not gone forward. (I do not oppose government funding to save lives.) No link has been found between ELF and cancer at normal conditions. No biological mechanism for cancer has been found. The epidemiology data does not show a consistent association between cancer and the measured magnetic fields. There are many confounding effects that can obscure very weak data.

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Dr. William Frazer, Chair of APS Panel on Public Affairs
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National Policy Statements

95.2 STATEMENT ON POWER LINE FIELDS AND
PUBLIC HEALTH

(Adopted by Council 23 April 1995)

Physicists are frequently asked to comment on the potential dangers of cancer from electromagnetic fields that emanate from common power lines and electrical appliances. While recognizing that the connection between power line fields and cancer is an area of continuing study by research workers in many disciplines in the United States and abroad, we believe that it is possible to make serveral observations based on the scientific evidence at this time. We also believe that, in the interest of making the best use of the finite resources available for environmental research and mitigation, it is important for professional organizations to comment on this issue.

The scientific literature and the reports of reviews by other panels show no consistent, significant link between cancer and power line fields. This literature includes epidemiological studies, research on biological systems, and analyses of theoretical interaction mechanisms. No plausible biophysical mechanisms for the systematic initiation or promotion of cancer by these power line fields have been identified. Furthermore, the preponderance of the epidemiological and biophysical/biological research findings have failed to substantiate those studies which have reported specific adverse health effects from exposure to such fields. While it is impossible to prove that no deleterious health effects occur from exposure to any environmental factor, it is necessary to demonstrate a consistent, significant, and causal relationship before one can conclude that such effects do occur. From this standpoint, the conjectures relating cancer to power line fields have not been scientifically substantiated.

These unsubstantiated claims, however, have generated fears of power lines in some communities, leading to expensive mitigation efforts, and, in some cases, to lengthy and divisive court proceedings. The costs of mitigation and litigation relating to the power line cancer connection have risen into the billions of dollars and threaten to go much higher. The diversion of these resources to eliminate a threat which has no persuasive scientific basis is disturbing to us. More serious environmental problems are neglected for lack of funding and public attention, and the burden of cost placed on the American public is incommensurate with the risk, if any.

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The Imprudence of "Prudent Avoidance"

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In 1988, shortly before he died, Andrei Sakharov commented on the fate of the earth. Interestingly enough, rather than comment on the hydrogen bombs that he co-invented, he stated: "... in fact, I am now inclined to regard the many-faceted ecological threat to our environment as our most serious long-term problem. I " Because I agree with this very long-term assessment, it is troubling to me to see environmental funds and political capital wasted on false threats. In particular, I am concerned that the quasi-legalistic concept of "prudent avoidance" is being used to chase the phantom risk of cancer caused by extremely low frequency (ELF) electromagnetic fields (ENE) from power lines. This needless chase costs some one to three billion dollars per year2,3 and unnecessarily frightens the public with " electrophobia. " The burden of these fiscal and emotional costs placed on the American public are incommensurate with the risk, if any, begin mitigated. This outcome is not a use of science for the public good.

What is Prudent Avoidance?

In the absence of any firm scientific demonstrated connection between ELF/EW and cancer, the concept of "Prudent Avoidance" has been invoked by many utility commissions (at least eleven by recent count) as a basis for promulgating regulations. Granger Morgan defines4 "prudent avoidance" as: "Prudence means exercising sound judgement in practical matters. It means being cautious, sensible, not rash in conduct." Morgan continues, prudent avoidance "is to try to keep people out of fields when that can be done at modest cost -- but not to go off the deep end with expensive controls which may not be beneficial. "

Prudent avoidance, as thus defined, might seem reasonable if one understood the nature and severity of the risk, which is not the case for the alleged EMF health hazard. From there Morgan moves towards suggesting the arbitrary spending of money without measurable benefits: "Utilities and utility regulators must consider both distribution systems and transmission systems. Activities that may warrant consideration at the distribution level include: paying greater attention to population distributions around facilities; incorporating more consideration of exposure management in maintenance and facility upgrade policies... making selected use of undergrounding..." At this point "prudent avoidance" becomes imprudent because it leads to an open-ended, unbounded approach to risk mitigation. It stimulates a fearful public to use the threat of litigation to force utilities and school boards to take steps to mitigate a phantom effect. These institutions have little incentive to risk litigation, as long as the costs of compliance will be covered by rate payers or tax payers.

Morgan's approach appears to be driven by his statement that "there is some significant chance that fields pose a modest public health risk, and not much chance that the risk to any one of us will be very big.4" In my analysis of Morgan's work, he seems to have placed great reliance on the very questionable work of Wertheimer and Leeper5 when he stated (1992) that "a series of epidemiological studies, including studies of childhood leukemia by Naorgan alludes4 to Thomas Kuhn's STRUCTURES OF SCIENTIFIC REVOLUTIONS by stating that "paradigm shifts" are affecting "scientific thinking about biological effects from electric and magnetic fields." It is premature to talk of paradigm shifts when the preponderance of the data does not demonstrate that there is a connection between cancer and these fields. Morgan is concerned that public perceptions may drive regulations rather than scientific fact. However, I conclude that it is his own papers that have strongly pushed the EMF-risk process away from science and toward irrationality. I agree with the critics of "prudent avoidance" who have call it "the abandonment of science, "the triumph of fear of the unknown over reason," and "being so vague as to be useless.4" Prudent avoidance is a delight for plaintiff lawyers since it is essentially a conclusion that the danger is probable.

The General Accounting Office (GAO) report7, ELECTROMAGNETIC FIELDS, acknowledges this misuse of science by cont the total economic costs of the [EW mitigation] activities described above now exceed $1 billion annually, with the promise of growing costs in the years to come..... If we were to value the reduction of a unit of EMF risk at comparable levels, the most that we could justify spending on EMF mitigation would be something in the neighborhood of $10 billion per year.... Recent examples include a town that moved several blocks of distribution lines underground at a cost of $20,000 per exposed person; a utility that rerouted an existing line around a school at a cost of $8.6 million, a new office complex that incorporated EMF exposure in its design at a cost of $100-200 per worker; and a number of firms that have installed ferrous shielding on office walls and floors to reduce magnetic field exposures from nearby power handling equipment at costs ranging up to $400 per square meter of office space.2"

The GAO study7 estimates the following costs for EW mitigation, which would not reduce the EMF from appliances from within the home.

-- $90,000/mile for delta design above-ground transmission lines to reduce magnetic fields by 45%,

-- $2 million/mile to bury transmission lines in fluid-filled steel pipes to reduce magnetic fields by 99%,

-- $1 billion to limit magnetic fields to 10 mG at edges of rights-of-way for planned new transmission lines,

-- $3-9 billion to reduce magnetic fields at homes where grounding systems are the dominant source,

-- $200 billion to bury transmission lines nationwide near homes with fields greater than I mG,

-- $250 billion to reduce average exposure to less than 2 mG from all transmission and distribution lines.

Allan Bromley, President Bush's Science Advisor, recently commented on an EMF study done by the Office of Science and Technology Policy: "It is safe, however, to conclude that the EMF risk issue will continue to be contentious and of immense potential economic importance; the current best estimate is that prior to 1993 it has cost the American public more than $23 billion to respond to public worries about EMF -- particularly in connection with the placement of high-voltage power lines. 3 "

Recently a law suit was filed against Houston Light and Power and the Electric Power Research Institute (EPRI) on behalf of eleven families with children suffering from cancer. The suit charges both the power company and EPRI with "fraudulent concealment of the carcinogenic nature of the fields that secretly and silently invaded their homes." To avoid such litigation and the associated unfavorable publicity, other institutions have decided to give in, rather than fight. For example: (a) The San Diego Gas and Electric Company cancelled a power plant upgrade and compromised on a 69-kV line. (b) Hawaiian Electric Industries, Inc., spent nearly $5 million to reroute and reconfigure power lines. (c) In the Nfill Valley School District, 4 classrooms, a day care center, and a part of the playground located near power lines have been closed. (d) The policy of prudent avoidance added about $500,000 to the construction costs of the World Bank Building, and this approach is now considered to be a model in this area. (e) The California Public Utility Commission has required the utilities to spend up to 4 percent of the cost of electrical projects to mitigate ENV. Thus, we see that the advocates of "prudent avoidance" are willing to spend large sums for mitigation efforts with no clear assessment of any benefits to be gained.

Evidence Bearing on Effects of ENV.

The scientific literature and the reports of review panels show no consistent, significant link between cancer and the ENIF from power lines8. This literature includes epidemiological studies, research on biological systems, and the analyses of theoretical mechanisms. This negative result is consistent with the implications of arguments which have been advanced that there can be no such link. The preponderance of the epidemiological and biophysical/biological research findings have failed to substantiate those studies that have reported specific adverse health effects from the exposure to 60-Hz EMFS. It is always possible that some minor carcinogenic connection might be found, but the present data do not establish that connection. To justify expenditures on mitigation, there should be some consistent, meaningful combination of the following factors: (a) a plausible coupling mechanism at the cellular level exists, (b) evidence that the coupling must produce consistent biochemical changes, (c) indications that the biochemical changes must be detrimental, (d) meaningful epidemiology data that determine the degree of danger, and finally, (e) application of upper-bound mitigation costs for ENT that are comparable to the mitigation costs for other dangers in society.

Epidemiology: The scientific panels that have reviewed the EMF epidemiology data have found8 them inconsistent and inconclusive. It is necessary when comparing the data to separate the results by cancer type. For example, consider8 the recent case of three studies of electrical workers and a recent study on non-electrical workers in Sweden. The 1993 California study reported no association of ENIF with either leukemia or brain cancer. The 1993 Canadian-French study reported an association with leukemia, and astrocytoma, out of the 32 cancer types studied, but this study suffers from problems of internal inconsistencies. The 1995 Savitz/Loomis study reported no association of ENV with leukemia, but they reported an association with brain cancer. The 1993 Swedish study reported an association with leukemia, but not with brain cancer. Thus, these four "best studies" report very contradictory results. It is very difficult to determine statistically relative risk factors of less than two for rare modes of death because of the many confounding factors such as economic status and chemical pollutants.

Biology and Biophysics Experiments: The scientific review panels, the review articles, and the research papers that we have reviewed8 do not claim a causal link between EMF and cancer. In addition, the review panels and review articles have pointed out that there is a continuing problem with replicating the experimental results on cells and animals.

Theoretical Mechanisms: No plausible biophysical mechanism for the systematic initiation or promotion of cancer by these extremely weak ENW's has been identified8. The lack of epidemiological evidence and experimental

evidence establishing a link between EN/fF and cancer is consistent with the biophysical calculations that rule out the carcinogenic effects because the thermal noise fields are larger than the fields from EMF. Since quantum mechanics, thermal noise fluctuations, and cancer promotion are all statistical effects, it is difficult to derive a proof that is a necessary and sufficient condition to preclude all cancer promotion. However, these fundamental calculations are a significant guide post to conclude that the EMF-cancer link, if any, should be extremely difficult to detect because its magnitude **is,** at most, very small.

Journalism: The number of newspaper stories on EMF rose from 233 in 1992 to 548 in 1993. The number of magazine stories rose from 101 in 1992 to 216 in 1993. The writings of Paul Brodeur, such as CURRENTS OF DEATH, have been followed with headlines of "Is My Electric Blanket Killing Me" to "Chilling Possibility: That A Power That Has Improved Life Could Also Destroy It" to "Warning: Electricity Can be Hazardous to Your Health." Even when an article is even-handed, its caption at the top read: "Steps to Protect Yourself from Danger -- Real and Potential." It is my conclusion that the science and relative risk methodology often undercut quality of journalism in a free and fear-prone society.

The statement issued by the Council of the American Physical Society (APS) (reprinted in the Comment section) addresses these concerns in more general terms.

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Fund for the Environment filed its California Public Records Act Request on 5/6/01 for the release of two suppressed Dept. of Health Services studies on the electro-magnetic fields.

One is on EMF risk evaluation and the other is entitled, "Policy Options in the Face of Possible Risk from Power Frequency Electric & Magnetic Fields (EMFs). They were finally released Friday, July 13 via publication on the Internet.

No press release had yet been issued. [DHS did so, after we sent ours]

As executive director of FFTE, I have been asked for my impressions of the studies' findings and conclusions.

These studies were due to be released on May 7 at a meeting of a distinguished panel of scientists. I was hoping to have the benefit of their evaluations then. I am glad to learn that the panel will finally be convened on August 7, and look forward to their discussions.

Having served on the CPUC's EMF Consensus Group ['91-'92] and as a member of the DHS Stakeholders Advisory Consultants panel ['94-'98] and having also participated in each of the SAC meetings, subsequently, I have been eagerly awaiting these reports.

They were expected to climax the outcome of ten years of EMF studies by the utilities, their research organization, EPRI, the Federal government's studies and EMF research efforts abroad.

As a journalist, [Los Angeles Times Consumer Advocate columnist '71-'78 and op-ed writer since] I have been asked what in these reports might be of particular interest, from the standpoint of news.

Three DHS' EMF reviewers are the reports' credentialed authors. They might say the greatest significance in these studies is that they found a probability of adverse health effects from elevated exposures to EMFs.

Previously, many researchers had acknowledged only the possibility of adverse health effects from such exposures. Despite this seeming departure, DHS used the word "possible" rather than "probable," in the title of its policy options document.

One aspect of the policy options report was of particular interest to me: Three times, within the 8 pages of the document, DHS specifically states, that they "will not be making any recommendations on policy at this time." After 10 years of study and having expended $10 million of Californian's ratepayer dollars, I had hoped DHS might state what, specifically, it thinks should be done. They say they are awaiting input from the public [Comments are due by Sept. 10] and they also note such decisions belong in the political arena.

The importance of these reports may be less in their very moderate content than in what their more than two-month suppression reveals about the Davis administration's M.O.

Its obsessive need to control all aspects of governmental proceedings, has been amply demonstrated, once again, by its delay in releasing the two, long-awaited, DHS/ EMF studies.

It seems evident that the governor wanted no possibility of the EMF issue being raised at this time. That's because he wants no doubts cast on his MOU with SCE and SDG&E. The MOU is for the Legislature to approve the State's purchase of these two utilities' power grids.

The probability or the possibility of latent liability, for ultimate damage to health from EMFs, might, indeed, be inferred by these reports.

Not mentioned in these studies is the recent public recognition of the hazardous nature of EMF's by Southern California Edison, In its filings with the CPUC, regarding the San Onofre Nuclear Generating Station,

Beginning last September, SCE reveals that it is now not only concerned with liability for nuclear radiation emissions but also for EMF emissions.

Had it not been for the conscientious and very public media attention to these "secret" studies we would undoubtedly still be awaiting their release.

We see a consistent need on the part of the governor, for total control. His assumption of emergency powers, as a means of concealing actions he has taken, with regard to the independent power generators, has been most egregious.

But it was not a first for him. Soon after his administration took office, his Secretary of Resources, an attorney, instituted appropriate legal action in a water matter. Governor Davis immediately chastised and humiliated her, publicly.

Subsequently, neither she, nor any other cabinet member has dared to initiate significant, needed action, without first getting the express permission of Davis. The Dept. of Health Services has learned that lesson well.

Davis' need to micro-manage has found him saying that the Legislature is there to implement his vision. This is a lesson also well learned by the CPUC which was said to be holding up the two EMF reports so it could first be briefed on their content.

But then, the CPUC claimed to be "too busy" to be briefed, causing further delay. Not incidentally, the CPUC signed a MOU with DHS in March of 1994 which said, in part, "Whereas [EMF research] Decision 93-11-013 states the desire of CPUC not to micro-manage this program . . ." This makes the CPUC's delaying of the reports more than suspect.

In light of the findings of the EMF studies, emphasis should now be placed on one of the best ways to minimize exposure to elevated levels of EMF. That is through the burying of power lines. The costs for doing so would be more than repaid in lifecycle costs.

These include the elimination of millions of dollars annually spent on trimming trees adjacent to and beneath power lines, minimizing wild fires associated with above ground powerlines, beneficial increases in property valuation, and therefore enhanced tax revenue to the localities.

The ancillary benefits would be to all Californians, in restoring the state's scenic, powerline-free, skyline.

Interestingly, a proceeding to investigate the expediting of undergrounding has been underway at the CPUC for a matter of years. It was instigated by a legislator, Dion Aroner. However, sufficient staff has yet to be assigned to this matter and the CPUC has held no meetings this year, on this proceeding. Fund for the Environment is a party in this matter,as well.

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6th September 2001

Dr Jack Collins
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Dear Dr Collins

Comments on "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances"

Comment 1

May I congratulate you and your colleagues on such a thoroughly professional and well thought out Evaluation. There is nothing like it in the UK or Europe and it should be a model for the authorities over here in their assessment of the EMF health effects. I thoroughly welcome the public release of this document. The incidence of certain cancers has been increasing significantly in recent years and it is our duty to ask the searching question as to why. It may be that exposure to power frequency magnetic fields has a fundamental role in the etiology of some cancers. This possibility deserves our serious attention.

Comment 2

I realise that you have been unable to address the possible adverse health effects of corona ions emitted from high voltage powerlines (transmission lines). Our work in this area is ongoing and I am enclosing two papers for your attention. The first is a copy of a poster session paper presented at the recent 23rd Annual Meeting of the Bioelectromagnetics Association in Minnesota in June of this year.

The second is a paper I have recently submitted to the journal Medical Hypotheses. This is a Risk Assessment which tries to put actual values on the number of cases of certain cancer and non- cancer illnesses associated specifically with high voltage powerlines in the UK. With regard to magnetic field effects my paper does not go nearly as far as your comprehensive risk assessment. I was interested that in your Evaluation you associate suicide with magnetic field exposures but regard the data on depression as inadequate to form a judgement. You will be aware that some of the key papers on this subject originate in the UK (notably by Dr Stephen Perry who sadly passed away just a few months ago). My advice from psychiatrists is that depression is a strong risk factor for suicide, so in this sense the two go hand in hand.

With regard to corona ion effects, you may be interested in the technical description I present in the paper since there has been widespread misconception about the physics behind the effects we have reported. You may also be interested in the number of cases of ill health in people living near high voltage powerlines in the UK (see table 3). The figures will undoubtedly have a bearing on a cost benefit analysis of burying high voltage powerlines.

I am not asking you to cite any of our work in any revision of your Document in the near future. I appreciate that the scientific community needs to wait until further research has been carried out and properly reported.

Comment 3

You may be aware that the UK National Radiological Protection Board exposure limit to magnetic fields is 1,600 ?T (16 G) and you are of course aware that the ICNIRP guideline is 100 ?T (1 G). In view of the introduction by the Swiss Government of a 1 ?T (10 mG) limit I wondered whether your Evaluation would express a view on this.

Once more may I congratulate you on your excellent Evaluation.

Yours sincerely

Professor Denis L Henshaw

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September 7, 2001

Dr. Raymond Neutra
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Dear Dr. Neutra:

Thank you for this opportunity to provide comments on the draft report, "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances."

These comments provide further information regarding your specific questions in addition to the joint answers from the California electric utilities. The enclosed comments specifically address your questions 1,3, 4, 5 and 7.

I hope you will find these comments constructive.

Sincerely,

Kent C. Jaffa

Kent C. Jaffa

September 2001

Comments on the CDHS Draft Risk Evaluation Report

Summary

The CDHS draft risk assessment substantially diverges from the conclusions of all previous EMF risk assessments by numerous scientific bodies. Is this because new data justifies such CDHS conclusions or are these conclusions a result of an inadequate evaluation? The evidence supports the later. The evaluation was hampered by only having three reviewers with similar expertise and does not adequately evaluate the literature. The CDHS draft assessment is not in agreement with the 1999 National Institute of Environmental Health Sciences report to the US Congress.[1] It is also not in agreement with reviews covering similar or more recent literature databases by the International Agency for Research on Cancer (IARC), the Advisory Group on Non-ionizing Radiation of the UK National Radiological Protection Board, or the Health Council of the Netherlands.[2-4] CDHS' divergence should be accompanied with substantial support, which is lacking in the draft risk assessment.

This divergence can be attributed to:

- **CDHS does not give appropriate weight to all of the EMF research data and focuses on their preconceived views.**
- **There is an over reliance on epidemiology while giving inadequate consideration to the animal, laboratory and mechanistic data.**
- **Proper consideration to bias is not given in their analyses of the epidemiology.**
- **There is a lack of attention to exposure metrics in their analyses of the epidemiology.**
- **Their consideration of animal and laboratory data is flawed.**

It is problematic to place undue weight on the epidemiology results since these results show only weak effects. There are very small numbers of high exposed people and the results may be partially explained by selection bias. No specific EMF exposure has been identified as well as other problems.

For example, there have been two major pooled analyses of the childhood leukemia studies.[5,6] One of these by Greenland *et al* is considered in the CDHS assessment. Greenland *et al* state "*In light of the above problems, the inconclusiveness of our results seems inescapable; resolution will have to await considerably more data on high electric and magnetic-field exposures, and possible sources of bias*."[5] Ahlbom *et al* state "*For the very small proportion (0.8%) of subjects with exposure above 0.4 mT, the data show a two-fold increase, which is unlikely to be due to random variability*." "*The explanation for the elevated risk estimate is unknown, but selection bias may have*

accounted for some of the increase."[6] CDHS seems to ignore the inconclusiveness of these findings including the small number of high exposures, and they interpret the childhood leukemia studies to be substantially more conclusive than the authors.

Lack of Attention to Participation Bias

As mentioned above, one of the reasons for the inconclusiveness of Greenland *et al*.'s findings is the important issue of bias that the draft too readily discounts. In the Greenland childhood leukemia study, the individual study with by far the largest number of cases of high exposures ($\geq 0.3\ \mu T$) is the study by Linet *et al*.[5] Authors of this study have shown that their reported elevated odds ratios for high exposures could very well be due to participation bias and confounding.[7] A substantial number of the childhood leukemia studies have low participation rates and this is a substantial factor in them as pointed out by a member of the SAP. In the second major pooled analysis, Ahlbom *et al* also agree that participation bias is a partial explanation for the elevated odds ratios at high exposures in their recent pooled analysis. Thus, selection bias is an important topic and the draft should pay stronger attention to it.

Exposure Issues in the Scandinavian Studies Limit Their Ability to Provide Insight on Participation Bias

When the topic of participation bias was raised by a member of the SAP, CDHS defended their position by arguing that the Scandinavian studies are not subject to participation bias. These generally elevated findings, however, may be subject to a different type of bias relating to their exposure metric of historical calculations. Thus, the Scandinavian studies do not necessarily argue against participation bias.

It has been shown that historical calculated fields may be flawed in the seminal Feychting and Ahlbom study.[8-10] The Swedish calculations have more misclassification error than their contemporary spot measurements, even though the spot measurements were made sixteen years after diagnosis. This demonstrates the poor accuracy of historical calculations in the Swedish study. Further, it has been demonstrated that their negative odds ratios for contemporary spot measurements appear to be more reliable than the positive results for historical calculations because calculation errors appear to operate differentially.

To the contrary, Feychting and Ahlbom have argued that exposure misclassification in their study is non-differential and historical calculations lead to reliable effect estimates even though calculations may have more total error than contemporary measurements.[11] Their argument is based on historical calculations having a high specificity with a low prevalence of high exposure. They imply that contemporary measurements are inferior since they have a lower specificity and were made on average sixteen years after diagnosis. Jaffa *et al* countered that the differences in specificity do not account for the difference in their results for their two metrics.[9] More recently, the journal *Epidemiology* will be publishing a letter by Jaffa giving further support that non-differential misclassification does not justify concluding that the Swedish positive results for historical calculations are better effect estimates than the negative effect estimates based on contemporary measurements.[10] Jaffa demonstrates that non-differential misclassification arguments should result in a more positive odds ratio for measurements than calculations if there is a true effect. The only other explanation is that some other parameter of the magnetic field is responsible for the association that is not captured by contemporary measurements or contemporary calculations and is not caused by historical power line current changes. It is difficult, if not impossible, to conceive of any magnetic field parameter that can meet these criteria. A copy of the peer-reviewed manuscript is included in Appendix A.

If the seminal Swedish study that introduced historical calculations is problematic, the other Scandinavian studies relying on calculations are also subject to question. Consequently, CDHS consideration of participation bias in measurement studies is insufficient and they over interpret the Scandinavian studies to justify this lack of attention to bias.

Lack of Attention to Exposure Metrics

Another reason why the results of the Greenland *et al* study are inconclusive is highlighted in the July 2001 issue of *Epidemiology*.[12, for further details see Appendix B] Perhaps, this criticism is best stated in the words of Greenland *et al*.

> *Those considerations also raise a serious criticism of our analysis, in that we pooled different magnetic field measures without demonstrating that all of the measures are comparable or combinable.…[O]ur criteria for choosing measures when we had a choice are not compelling … and one could reasonably argue in favor of other choices… We caution … that other choices could lead to very different degrees of variation; our results may not even be typical of what would be seen upon trying all defensible choices …*[5]

A lack of attention to the differences between metrics in the CDHS draft report is also a serious criticism of their analyses.

This lack of attention can be seen in the report's tables. For example, the report shows an odds ratio of 1.37 for childhood leukemia (Table 8.1.3, p. 76-79) and 3.9 for childhood brain tumors (Table 10.1.1, p. 133) for the Tomenius study. Attention to exposure metrics presents a much different perspective as the Tomenius study actually shows higher effect estimates at distances greater than 50 meters compared to under 50 meters.[13] Tomenius' published results really argue against EMF causation.

A similar argument can be made with the Schreiber study on residential female breast cancer. CDHS reports this study shows an odds ratio of 1.00 (Table 11.1.1, p 153), however it actually reports a SMR of 0.96 within 100 m of transmission facilities and 1.28 at distances greater than 100 m.

For occupational female breast cancer, CDHS reports that the Loomis study shows a risk of 1.38 (Table 11.1.2, p. 154). It does not, however, consider that Loomis does not discriminate between low, medium and high occupational exposures.[15] Loomis reports one of the highest effect estimates was for electrical engineers (RR=1.73). This argues against causation and for bias, as electrical engineers in general have similar exposures to office workers based on measurements. Even amongst the electric utility industry where engineers would seem to have higher exposure than engineers in general, engineers were not considered as an exposed occupation in the Savitz electric utility workers study based on exposure measurements.[16-17].

On the other hand, Cantor's study essentially consists of the same population with one additional year of data and this study breaks down exposure into low, medium and high exposure categories.[18] Cantor found an odds ratio of 0.97 for high exposure. A more comprehensive look at all of the studies from a dose response perspective should be included in the evaluation. This is an important consideration when interpreting the epidemiology.

It is also noteworthy that the Tomenius childhood leukemia study actually reports an odds ratio of 0.3.[13] CDHS does not state where the 1.37 odds ratio comes from in Table 8.1.3. It is either an error or an unpublished reanalysis of the original data that should be documented.

In addition, there is really no consistent pattern between effect estimates and increased metric accuracy. This observation is not examined in the report and it should not be used solely to justify the lack of specificity or precision in the consideration of metrics. While this may be used as an argument for their "mixture" of EMF parameters, it is also a strong argument for bias and a lack of specificity in the overall consideration of disease causation. It is no excuse for ignoring the information that can be gleaned by examining the implications of the metric-specific results.

In this context, it is noteworthy that the CDHS draft evaluation selectively reports results from the childhood leukemia studies. To illustrate a few examples, Table 8.1.3 (p. 76-79) shows Tomenius results for measurements, but not for proximity. It reports Feychting and Ahlbom's results for calculated fields, but not for proximity or measurements. It includes Tynes' results for calculations, but not for measurements. It includes Linet's results for measurements and wire codes, but not for proximity. It does not report the wire code results for McBride and Green or their personal dosimetry and other metrics, but it does include their spot measurements. Without a good table of results, it is impossible to do an adequate job of evaluating the epidemiology studies.

As metric accuracy has increased, the effect estimates have not generally increased and have become more subject to questions of bias. The fact that wire codes and measurements are correlated but show similar and contrasting effect estimates in different studies is supportive of bias as well as the lack of a clear dose response. Personal dosimetry close to the time of diagnosis does not show a stronger effect than 24-hr measurements, historical calculations or spot measurements made several years after diagnosis in the Savitz or London studies. A lack of metric specificity is an argument against causation. CDHS should revise their risk assessment to account for the implications of exposure metrics.

Consideration of Animal and Laboratory Data

Other examples of over interpretation of the epidemiology could also be provided for other disease endpoints. Nevertheless, in these circumstances, more weight should be placed on the lack of animal, laboratory and mechanistic support. The lack of this support is a major consideration in all other scientific panel reviews.

It is not necessary for me to provide support for this view as it can be found in the numerous other scientific review panels. I would like, however, to provide some additional information on the hen-house studies as I believe that it provides an important perspective.

Recently at a SAP meeting, CDHS defended their draft miscarriage conclusions by citing the hen-house studies. Their view is inconsistent with the group of scientists that NIEHS assembled to discuss the reproductive clinical and *in vivo* data in 1997 in which two CDHS personnel participated and expressed their views.[19] This group found and agreed unanimously that the chick egg studies were equivocal and that chick assays are not good for human risk assessment. In addition, they found and agreed that the mammalian studies were between no-effect and equivocal. The science has not changed significantly from these findings and they are more typical of the scientific community's views than CDHS' views. A copy of this NIEHS report is included in Appendix C.

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Appendix A

Attached is an in press copy of the letter by Jaffa regarding non-differential misclassification in the Feychting and Ahlbom childhood leukemia study.[10] This letter is scheduled for publication in the November 2001 issue of *Epidemiology* and is included in this appendix with their approval.

The authors reply:

Mezei and Kheifets show that the principles we relied on in our discussion of non-differential misclassification in the Feychting and Ahlbom study are not correct for all situations.[1-3] Sometimes a less accurate metric can result in the best effect estimate and there can be a greater difference between effect estimates of two metrics when there is better agreement between metrics. Their observations do not, however, appear to apply to the Swedish EMF study of childhood leukemia.

To demonstrate if non-differential misclassification can explain the Swedish results, the characteristics of Mezei and Kheifets' examples need to be similar to those of the Swedish study shown in Table 1. Non-differential misclassification must result in historical average calculated fields (HACFs) and contemporary spot measurements (CSMs) effect estimates both above and less than one in the same dataset, respectively. Their examples do not meet these criteria.

To determine if non-differential misclassification can actually cause this behavior in the Swedish study, Figure 1 was developed to show relations among specificity, sensitivity and effect estimates. The boundary between effect estimates (above and below 1.0) occurs where the sum of the sensitivity and specificity is 100%. This is valid for any dataset with a dichotomous cutpoint, assuming the "truth" is greater than one. The boundary is also independent of the control prevalence of high exposure, the case-control ratio and the positive value of the "true" effect.

Lines representing total error as a function of specificity and sensitivity are also plotted in Figure 1. These error lines are for a "true" effect of 5.3, a case-control ratio of 0.072 and a control prevalence of high exposure of 7.6% (ballpark values for the houses-only dataset). The total error lines are a function of the "true" effect, case-control ratio and control prevalence of high exposure. Error lines representative of other Swedish datasets or the Mezei and Kheifets examples would show some parallel shifts, but the same general trend occurs resulting in similar conclusions.

Estimates of the HACF parameters (Table 2) can then be plotted on Figure 1 to determine the likelihood of a non-differential misclassification explanation. With a total error less than 10% (representative of the combined and houses datasets), Figure 1 shows it is unlikely for non-differential misclassification to result in effect estimates that are both greater and less than 1.0 by varying the specificity and sensitivity. With greater error, CSMs would require substantially more total error and a lower sensitivity, compared to HACFs, to result in an effect estimate below 1.0. We have previously demonstrated that CSMs do not have substantially more total error. The total CSM errors are either similar to or less than HACF errors, depending upon the dataset.[1] CSMs should also have higher sensitivity since calculations underestimate measurements. Thus, it is unlikely that non-differential misclassification can explain the Swedish results. Non-differential misclassification is also an unlikely explanation for the negative CSM effect estimate since the error and sensitivity move the CSM effect estimate further from the boundary compared with HACF.

This discussion is based on the relative error between metrics and assumes there is no attribute of electromagnetic fields (EMFs) other than historical power line changes that is captured better by HACF than CSM. It is possible that there is another EMF attribute such that (1) the relative accuracy between CSM and

TABLE 1. Characteristics of the Swedish dataset

Dataset	Overall Metric Accuracy	Effect Estimates
Combined	CSM > HACF	CSM < 1.0, HACF > 1.0
Houses only	CSM = HACF	CSM = 1.0, HACF > > 1.0
Apartments only	CSM > > HACF	CSM < 1.0, HACF = 1.0

Note: CSM are contemporary spot measurements and HACF are historical average calculated fields.
Overall accuracy is the total percentage of misclassification including both exposed and unexposed. ">>" means much larger than ">," not much much greater.
Based on references 1 and 4.

TABLE 2. Estimates of the Overall Error, Specificity and Sensitivity of HACF Based on a 0.2 μT Dichotomous Cut-point and the Comparison of Contemporary Spot Measurements and Calculations in the Feychting and Ahlbom study

Dataset	Overall Error %	HACF Specificity %	HACF Sensitivity %
Combined	6.5	98.6	62.8
Houses only	9.2	98.6	84.8
Apartments	14.3	98.4	57.6

Derived from Tables 3.6, 3.8 and 3.10 of reference 4. The actual overall error, specificity and sensitivity are unknown, as historical measurements are unavailable.

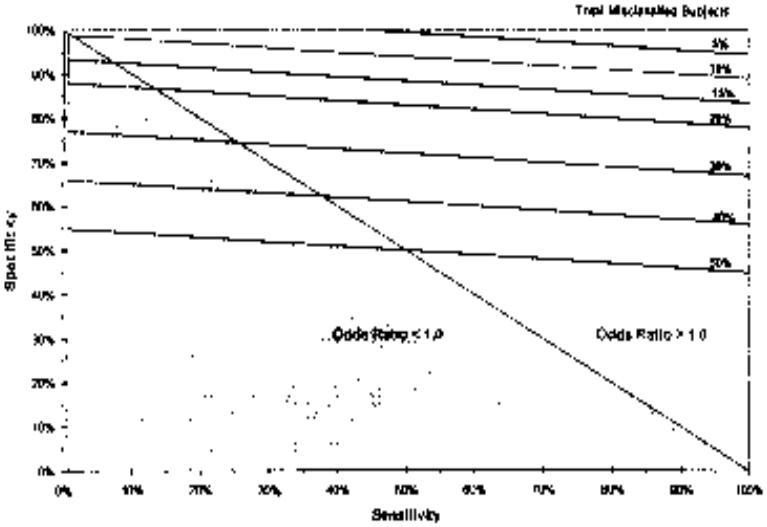

FIGURE 1. Effects of non-differential misclassification on the odds ratios of a dichotomous distribution of cases and controls

and is not caused by

HACF is not meaningful and (2) the resulting error is much larger in CSM than HACF. There is, however, little support for such an EMF attribute that is not related to CSM contemporary calculations or historical power line changes. Thus, justification is lacking for concluding that the HACF positive effect estimate is a better estimate of the truth than the CSM negative effect estimate in the Swedish study.

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Appendix B

As mentioned in this report, the pooled analysis by *Greenland et al* is subject to a serious criticism -- not demonstrating that the metrics they chose to pool are comparable or combinable so that it is unknown how representative their pooled results are. This issue was highlighted in reference [12]. Readers should be aware that Greenland *et al* took exception to reference [12], although they agreed that their results are inconclusive. [20]

Attached is a memorandum by Jaffa for those wanting further information regarding his letter. [12]

Kent Jaffa
July 2001

Comments on the Letter by Greenland et al.
Published in *Epidemiology,* July 2001

I viewed the Greenland et al. results as unreliable since the pooled data was not demonstrated to be reliable. In their November 2000 paper, they recognized that this was a serious criticism of their study and stated that "we pooled different magnetic field measures without demonstrating that all of the measures are comparable or combinable." "...[O]ur criteria for choosing measures when we had a choice are not compelling ... and one could reasonable argue in favor of other choices..." "We caution ... that other choices could lead to very different degrees of variation; our results may not even be typical of what would be seen upon trying all defensible choices..." Thus, under these circumstances, I don't think that it is an unreasonable view that their statistical analyses do not overcome the deficiencies of not assigning exposure as uniformly as possible or thoroughly investigating the implications of mixing metrics. Any reported finding, whether conclusive or inconclusive, is inconclusive in my opinion if the representativeness of the fundamental exposure assignment is unknown. I don't believe this position is based on "errors of logic, statistics and fact."

I was also disappointed that I was characterized as misrepresenting the Feychting and Ahlbom study. My letter only states that "It has been demonstrated that [their] spot measurements **may** be more accurate predictors of historical exposures than their calculations." This is not a misrepresentation as my paper does demonstrate the possibility of this whether or not others may question it. Due to the space limitations, I felt that it was only appropriate to cite the papers demonstrating this possibility and so I did not cite Feychting and Ahlbom's response. I wasn't trying to hide Feychting and Ahlbom's commentary or misrepresent their Swedish study. One of my citations cites the Swedish response and Greenland's paper does also.

Their complaint is also overstated that I did not mention that the Swedish measurement population is a subset of their calculation population. This is something that I discussed in my cited paper on the Swedish study. Feychting and Ahlbom reported that they viewed that their measurement population was a representative population conflicting with Greenland's view. Further, I showed how, if anything, the measurement population could have under represented the difference between measurements and calculations. Neither of us felt that it was appropriate to have a comprehensive discussion on this point. Since a comprehensive discussion was beyond the scope, I did not discuss it while they chose to only tell one portion. It is noteworthy that Feychting and Ahlbom's controls for leukemia and calculated fields are not a completely non-random selection either. Their controls were chosen to match all cancer cases as opposed to just leukemia cases.

Their view that the relevant exposure is unknown is not justification for ignoring a metric from one study while using the same metric for other studies. This really argues against their pooling assignment.

Greenland *et al* fault me for not citing the pooled analysis by Ahlbom et at., however I did cite this paper in my original submission (although in a different context), but I left it out when the journal asked me to substantially reduce the size of my manuscript.

Perhaps, the most disappointing statement was their claim that I misrepresented the views of Mary McBride. The only view that I stated of hers is that she considered her contemporaneous metric to be superior to her lifetime predicted metric. She told me this in a telephone conversation. Nevertheless, I wanted to be extra careful that I did not misrepresent her view and I sent her my original manuscript prior to submission so she could see the context in

which it was used and review it for accuracy. She responded with no comments thanking me for the opportunity. I don't know what more I could have done to insure that I didn't misrepresent her view if I really did.

I was also surprised by the manner in which they made their statements regarding laboratory data. They could have made their statement without implying that I misrepresented the laboratory studies. Dearth means a scarcity, lack of, paucity (smallness in number). Dearth does not mean that there are no positive results, but there certainly is a lack of laboratory support when looking at all of the research. This is how NIEHS, their reference 7 that they quoted to support their position, summed up the laboratory data. "Virtually all of the laboratory evidence in animals and humans and most of the mechanistic work done in cells fail to support a causal relationship between exposure to ELF-EMF at environmental levels and changes in biological function or disease status. The lack of consistent, positive findings in animal or mechanistic studies weakens the belief that this association is actually due to ELF-EMF, but it cannot completely discount the epidemiological findings." Dearth is consistent with their letter as well as mine.

In summary, I do not believe that my letter purposely misrepresents anything. I went the extra mile with Mary McBride. I even extended the professional courtesy of sending Greenland *et al* a copy of my original submittal at the time of submission giving them an opportunity to contact me and suggest changes prior to publication, something that they did not reciprocate with me. Before submitting my original Swedish paper, I also sent copies to Feychting, Ahlbom and Kaune giving them the opportunity to comment prior to submittal. I am well aware that some, not implying Greenland et al., may not objectively consider my views because I am not an established epidemiologist.

It is unfortunate that their letter did not concentrate on the real issue raised in my letter, the case/control assignment to a common exposure scale. The Feycthing and Ahlbom and McBride examples illustrate that different metrics are not equivalent. Other illustrations could have been shown when eight different metrics were used to represent twelve studies. As acknowledged in their original paper, this is a serious criticism and their response did not contain sufficient information to argue otherwise. It is still my opinion that this makes their study inconclusive independent of their results or the number of subjects in the high exposure categories. Their first preliminary results that they presented at a BEMS meeting showed no association for high fields, only at intermediate exposures. Upon further analysis, they appear to have decided that it was not appropriate to mix wire codes, measurements and calculations when pooling. While they separated their eight measurement and calculation metrics from wire codes in their published paper, questions still remain regarding their pooling exposure assignment of cases and controls.

Appendix C

Attached is a copy of the 1997 NIEHS breakout group report for clinical and *in vivo* laboratory findings pertaining to reproduction and development.[19]

EMF Science Review Symposium

Breakout Group Reports

for

Clinical and *In Vivo*

Laboratory Findings

Reproduction and Development

Speaker: Jukka Juutilainen, Ph.D.

Facilitator: Neil Chemoff, Ph.D.

Rapporteurs: Bernadette Ryan, Ph.D. and Mary Ellen O'Connor, Ph.D.

Introduction

In order to evaluate the potential for EMF effects on human health, the Breakout Group (BOG) was charged with the task of reviewing the literature and assessing the data based on the following criteria:

Quality of the data

Magnitude of the effect

Consistency across studies

Specificity of the finding

Clarity of cause-effect relationship

Strength of dose-response relationship

Mechanistic underpinning to findings

Analogy to other agents

At the onset of the discussion, the BOG decided that understanding of mechanistic underpinnings was not essential for determination of an agent as a teratogen. The BOG agreed that the weight of evidence for a potential hazard with respect to development should be ranked with human data as the most relevant followed by *in vivo* mammalian data and then non-mammalian chick or *in vitro* data.

The main question the group attempted to answer was whether the *in vivo* data provide conclusive support for or against a relationship between EMF exposure and reproduction and/or development. The existing data base was reviewed by discussing chick embryo studies, followed by those done with mammalian species. All studies on chick embryos utilized a magnetic field (MF), but there were both electric field (EF) and MF exposures to consider in mammals. For purposes of this report the chick embryo is discussed under the category of *in vivo.*

Over 120 studies were identified, but many had inadequate controls, inadequate control of or documentation of exposure, etc. Fifteen papers were distributed to the BOG based upon their significance for the EMF area and/or their recent publication date. Two of the papers were recent reviews (2, 3) which summarized pre- 1 993 studies. These were included to provide a more extensive reference base and to help participants put the selected papers in perspective. The review papers were not evaluated, but the speaker, facilitator, and both rapporteurs felt that they adequately summarized the available literature and were objective in presenting the parameters and reported conclusions. An additional review paper that reached generally similar conclusions as the others was provided during the meeting **(8).**

Discussion by the Breakout Group

The discussion addressed EF and MF studies on both avian and mammalian systems.

A. Chick Embryo

The BOG reviewed four studies including the Berman *et al.* (1) study done in six different laboratories (the "Henhouse study"). The papers reviewed were representative of the larger body of chick embryo literature. Of the studies discussed, two presented some positive effects (1, 17), one found no exposure related adverse effects (4), and one was a statistical re-analysis of the "Henhouse study" (6) retaining the overall positive effect.

The "Henhouse study" (1) used a unipolar, pulse magnetic field (500-μsec, pulse duration, 100 pps, 1 μT peak density, 2 μsec rise and fall times) was applied to exposed eggs during 48 h of incubation. Identical equipment was used in each laboratory and the individual investigators attempted to follow similar procedures. Differences between laboratories existed in terms of strain of animal, feed source, ambient fields, and handling of the eggs prior to exposure. The combined studies show a statistically significant positive effect for exposure and increased incidence of anomalies, and for the interaction of incidence of anomalies and laboratory. The BOG concluded that the individual studies were well-conducted. Two labs reported significant effects of exposure, three labs reported non-significant findings in the same direction, and one lab reported non-significant findings in the opposite direction with the control group having more anomalies than the exposed. The BOG noted that the proportion of abnormal embryos in the controls was high and variable (6% to 30%). The re-analysis of the data indicated that interlaboratory differences contributed an amount of variation comparable to the exposure effect itself. The BOG also noted that variability in control abnormalities might have been attributable to different egg collection and handling procedures used by suppliers in the various countries. Criteria for assessment of the embryos were not clear and several of the anomalies might have been reflective of growth retardation rather than actual defects. This is an important distinction, because small differences in growth may have no biological significance if the embryos achieve a normal stage of development at a later point.

Cox *et al* (4) used 50 Hz at 10 μT and found no significant exposure related effects. Ubeda *et al. (I* 7) used 100 Hz at 1 μT peak-to-peak amplitude and 500 [μsec pulse duration with two different pulse wave forms of 85 μsec (PMF-A) or 2.1 [μsec (PMF-B). The PMF-B exposure was closer to the HenHouse project's exposure conditions. They report an excess of anomalies with both waveforms. However the incidence of anomalies was significant only for the PMF-B group, which showed an increase in early embryonic death. The BOG noted that many of the previous concerns regarding possible inter-laboratory differences and inconsistency of the results noted for the "Henhouse study" also apply to these two reports as well as to the body of additional chick embryo studies.

On the basis of the studies reviewed, the BOG concluded that given the problems identified above, it is impossible to reach definitive conclusions concerning the effects of EMF on avian development. There was some discussion concerning wording to describe the BOG's conclusions as to the significance of the data. In the

time available, it was decided that three categories of certainty or uncertainty regarding the biological significance of the data would be used: "no effect," "clear effect," and "equivocal." The latter term indicated uncertainties concerning the data sets that might be due to a wide variety of factors including reproducibility between studies and/or laboratories, the nature of the criteria evaluated, the robustness of the effect, or possible influences due to different exposure parameters used in the various laboratories. After evaluating the avian studies, the BOG simply voted as to the appropriate description of the data, and it was overwhelmingly considered to be equivocal due to any or all of the factors used to define "equivocal". The BOG did recognize the utility of the chick embryo assay for studying mechanisms underlying established significant effects.

B. Mammalian Electric Fields (EF)

A number of studies examining the developmental effects of EF in the range of 10- *1 5* 0 kV/m at 60 Hz have been performed (I 2, and reviewed in 2 and 3). The preponderance of the studies published (greater than 10) showed no treatment-related adverse effects. Two studies had positive effects, but they also had possible confounders. A third large, multi-generation swine study showed an inconsistent response across generations. It was also possibly confounded by disease outbreak and treatment with antibiotics. This large swine study could not be repeated, but a multi-generation rat study (I 2) was performed in an attempt to mimic the swine study. In the rat study, no consistent treatment-related effects were observed.

The BOG concluded that the weight of the evidence clearly indicates no association between EF and adverse developmental effects.

C. General Magnetic Fields (MF) Mammalian Discussion: Introduction

The BOG evaluated all of the MF mammalian studies based on the number of "+" and "-" conclusions reported in the tables contained in the review paper by Huuskonen *et al* (8). This discussion did not include detailed evaluations on magnitude of responses reported, field types and strengths, and the actual fetal effects observed. The BOG reached a decision to summarize sawtooth (20 kHz VDT) and sinusoidal fields (50 kHz) separately.

D. Magnetic Fields (18-20 kHz)

Compared to the studies on EF, there is a larger body of literature on MF. The BOG had eight papers to review in this general area before the meeting (7, 9, 10, 13, 14, 16, 18, and 19). Three of the papers (9, 16, and 18) included were studies in which pregnant mice were exposed to fields (18 kHz or 20 kHz frequencies) similar to those generated by video display terminals (VDTs). The first of the studies (I 6) used several intensities of magnetic field (5.7 to 66 μT) and reported no alterations in maternal or fetal parameters at any of the field intensities used. The minor skeletal variations that were observed were considered to be normal background fluctuation in teratological evaluation. A large, carefully controlled study (18**)** was done at 20 kHz with intensities of 3.6, 17 and 200 μT. There were no significant differences between the exposed groups and controls for any developmental endpoints. A study discussed by the BOG (although not included in the original set of papers) reported a significant in resorptions associated with exposure to 15 μT at 20 kHz (5). It was pointed out during the discussion that this conclusion was somewhat questionable, because the exposed groups also had increased implantation sites and there were no differences in the average litter sizes at term when control and treated groups were compared. A recent study (9) attempting to replicate the increased resorptions seen previously (5) did not report any statistically significant changes in resorption associated with exposure. Considered as a whole, the body of data for 20 kHz exposures was considered by the BOG to be equivocal. A minority of the BOG felt, however, that the studies indicated no treatment-related effects given the size and quality of one of the studies (18**)** coupled with the interpretation and replication problems of the studies investigating resorption incidence.

E. MF 50-60 Hz

Of the six papers reviewed by the BOG (7, 9, 10, 13, 14, and 19), five indicated a lack of developmental effects associated with EMF. Two of the studies contained large numbers of animals (13, 14) and showed no treatment-

related effects. One study had statistically significant increases in minor skeletal anomalies/variations (7) which are commonly observed across all groups in teratology bioassays.

For the sinusoidal studies, one was positive at 200-300 Gauss (20 - 30 mT) (11,15), while another "+" had an increase in skeletal variations and implants at 12.6 μT (9). The first two papers were not in the BOG's packet and were not critically evaluated.

Finally, the BOG briefly discussed reproductive effects from an unpublished National Toxicology Program continuous breeding study performed by Ryan (unpublished data). The data did not indicate any adverse effect on reproduction. One statistically significant finding, occurring only in one generation and one sex, was increased adrenal weight. This finding was considered due to chance and of no biological significance. Some concerns were expressed about the paucity of data regarding estrus cyclicity and puberty onset. No conclusions were reached because the BOG did not have published papers to review. The opinion of the BOG was equally split between "no-effect" and an "equivocal effect."

Upon review of the report, it was noted that a paper on this topic was distributed during the session; however, the article was only available in Russian (20), so the BOG could not directly evaluate the study.

Other Issues Discussed in the Breakout Group

During the course of discussion, a number of general comments and questions were raised by the BOG. These included issues regarding data interpretation in teratology bioassays such as: What is abnormal? Is resorption considered a teratogenic effect? Can something be considered an anomaly when it occurs within normal spontaneous rates? What are the definitions of anomalies, variations, and minor malformations? What are the criteria for repeatability? It became clear that these issues and questions were long-standing and no simple answers exist. These issues are still debated within the area of teratology and an understanding of a scientist's criteria for many of these terms is essential for interpretation of his/her conclusions. Another comment that was made concerned the importance of doing studies with species/strains for which historical control data exist.

Additional observations were that in many of the studies in this area, control data seem to be quite variable, significant effects often are seen when the control values are low, and the effects noted after EMF exposure might be of biological interest but appear to lack relevance for human health.

Summary and Recommendations

1. Do the in vivo data provide conclusive support for or against a relationship between EMF exposure and reproduction and/or development?

Chick Egg Magnetic Field

Unanimous that the findings are equivocal

Mammalian Electric Fields

Preponderance of studies show no effect

Two positive studies were questionable due to confounders

Possible confounders in large swine study

Mammalian Magnetic Field

50- 60-Hz: BOG split between no-effect and equivocal-effect

20 kHz: BOG split between no-effect and equivocal effect

2. Are the experimental findings consistent across all studies? Are there specific EMF exposure parameters?

Chick egg

Consistency - Within certain laboratories but not between laboratories

Limited data set to compare exposure parameters - no effects < 1 μT

Mammalian Electric Field
Consistency - Yes for no effect
Parameters: -10 - 150 kV/m at 60 Hz

Mammalian Magnetic Field 50-60 Hz

Consistency - No

Parameters: 0.02 - 30 mT

Mammalian Magnetic Field 20 kHz

Consistency- No

Parameters: sawtooth pulse at 15 and 66 @T

3. Is there evidence of a dose-response relationship?

Dose-response generally not observed

Findings not consistently replicated
4. Are there complementary data available from biomarkers to support the experimental findings with respect to EMF effects on reproduction and/or development?

Not addressed

5. Are there gender-specific EMF effects?

No conclusion, because the BOG did not have relevant published papers to review.

6 Is there an explanation for any apparent differences in EMF exposure effects in mammalian versus non-mammalian species?

Chick embryo generally more sensitive to external agents than *in vivo* mammalian embryos

7. Is there any experimental evidence that in utero EMF exposure can cause long-term development effects

Not addressed

8. Do the in vivo data support epidemiological and/or in vitro findings with respect to EMF effects on reproduction and/or development?

Not addressed

9. How strong is the biological rationale in support of a relationship between EMF exposure and effects on reproduction and/or development?

Biological rationale unknown because of equivocal findings

10. What experimental evidence is lacking, if any, for conclusive support regarding EMF exposure effects on reproduction and/or development?

Replication, dose-response, identification of possible confounds and modifying factors, identification of appropriate exposure metrics

11. Are there short-term studies that could be undertaken that would resolve the discrepancies?

No

12. Briefly address the "Henhouse " studies. Address the quality of the studies. Is the experimental model relevant for studying mammalian reproduction

- Individual studies were well conducted
- Two laboratories reported significant positive findings
- Three laboratories reported non-significant positive findings
- One laboratory reported non-significant negative finding
- Abnormality in control high and variable
- Possible confounders (e.g., species differences, egg handling and storage procedures, geomagnetic field conditions, etc.)
- Inter-laboratory differences as large as exposure effect
- Chick assay good for examination of mechanisms but not for human risk assessment

13. How might information from the Hen House studies be used for understanding effects of EMF?

Chick embryo is appropriate for studying mechanisms of established significant effects.

Future Directions

Additional large-scale animal studies are not needed at this time. Once strong definitive studies have been done, if these do not provide a clear answer, the issue should be tabled until there is new insight from hypothesis-driven basic research.

Additional basic research on mechanisms is needed to identify appropriate metrics.

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Acknowledgments

The NIEHS EMF RAPID Program wishes to thank all attendees of this third EMF Science Review Symposium for their participation in the breakout group discussions and for their contributions to the breakout group reports.

The Program appreciates the time and effort provided by the speakers in the breakout sessions. Also the Program thanks the facilitators, rapporteurs, and speakers for the drafting of these reports. In addition, we would like to acknowledge the technical assistance of Judy Fleming, Katrina Hauser, Karla Solomon Jones, and Stephanie Russell in preparing and compiling materials for the breakout groups and for help in preparing this booklet.

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14 January 2002

Dear Raymond

I have read your extensive document with great interest. Below I will attempt to answer the questions you posed to the reviewers. Specific comments on the document are enclosed.

1. My prior degree of confidence is very low (but I would not say it is vanishingly small). I do not believe that average fields of 3–4 mG can produce health effects. Nor do I believe that we can distinguish between 1 and 4 mG with existing methodology. However, that does not mean that I am inclined to discount the observed association with childhood leukemia based on simplified biophysical calculations. Rather, I believe we have a clue (currently best captured by average fields above 3–4 mG) that needs to be understood.

   Ideally, biophysicists will provide us with testable hypotheses, but even lacking these, their arguments are important in checking sanity of proposed theories.

2. I believe eliciting prior degrees of confidence is a tricky business and I do not think informal solicitations, such as your question, provide useful and comparable information.

3. While lack of mechanistic understanding tremendously hinders progress in this field, for me it is not a sufficient argument.

4. I think you are giving too much weight to the breast cancer promotion studies (especially the Soviet results). Even more problematic, in my opinion, are the chick embryo experiments. I find that they provide no information at all.

   In the NIEHS evaluation, negative animal evidence pulled my assessment of the epidemiologic evidence down a bit. At IARC, I was persuaded by the argument that the animal data is not as negative as previously thought (based on the NTP experiment), although I did not think that there was enough evidence to classify the animal data as limited. Overall, I think high-quality and relevant animal data can be used to pull down epidemiologic evidence somewhat, but I do not see how it can **increase** the degree of confidence.

/…

5. I find your question too general, size of relative risk is only but one consideration. Risks between 1 and 2 should be interpreted with caution, as they can be easily explained by bias and confounding. Nevertheless, for childhood leukemia, I do not think the existing evidence can be ignored, until we have specific evidence for bias or confounding, or combination of both.

6. Lack of specificity does pull down my confidence a bit, and certainly does not increase it.

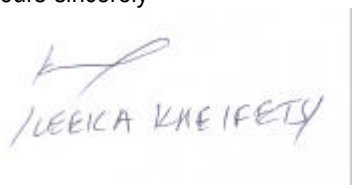
7. As you recall, I am not in favour of laying down “pro” and “con” arguments as you proposed.

8. I suggest changing “possible >50%” to “probable” and “possible <51%” to “not very probable.”

I hope these answers are useful. My personal assessment of the evidence is much closer to the IARC evaluation than to that of the three reviewers in your document.

Please let me know if any of the above requires clarification.

Yours sincerely



Dr Leeka Kheifets

Head, Radiation Program

Occupational and Environmental Health

Protection of the Human Environment

Comments on the California Department of Health Services Draft EMF Risk Evaluation and Policy Options Summary

EMF Risk Evaluation

Specific Comments

Page #	Table#	Line # or Comment # in Table	Comments
Entire document			Units for magnetic field measurements should be consistent throughout the document—either mG or μT.
5		1–3	Is it really true that the Stakeholders' Advisory Consultants (and not PUC) asked CDHS to make a determination as to the hazard of EMF?
5		15–17	A discussion of the applicability of IARC classification to non-cancer outcomes needs to be added.
6		6–7	*Two* laboratories in the Soviet Union reported copromotional effects of magnetic fields on breast tumor occurrence in rats? I am aware of study by Beniashvilli. Need references throughout.
6		42–44	In what studies were transient changes in magnetic fields evaluated? If an Enertech study is being referred to, any such evaluation was only circumstantial.
10	1		Table 1 is not consistent with the epidemiologic sections.
10	1	Row 3, column 3, line 1	For adult brain the IARC classification of Reviewer 1 should be "carcinogen." (see p.125)
11	1	Row 4, column 3, line 2	The IARC classification for ALS for Reviewer 2 can be read as "probable" in the text (see p.242), please clarify.
13		29	Adult brain cancer is erroneously included in the "Possible Cause" category. It should be in "Possible Carcinogen".
15		23	Should *visibility* perhaps be replaced with *plausibility* in this sentence?
16		12–14	Because other metrics have not been examined (as e.g., the metric you used in the study of spontaneous abortion), it is not possible to state that average magnetic field exposure represents a sufficient summary exposure metric.
16		44–50	Do these numbers refer to cases of cancer in California? Per year?
16		52–54	How this conclusion was arrived at is not clear. Was a 4% attributable risk assumed? Assuming 4% is problematic owing to the difference in risk and exposure for children and adults. For which cancers was the population burden summarized?
17	10.1	Row 2, column 3	Strictly speaking, only acute myocardial infarction from occupational exposure and childhood leukemia would be "of regulatory interest"

		and row 4, column 3	based on the information provided.
64		41	Which animal studies show an effect at μT levels? What level specifically?
64		59	The "alternative hypothesis" needs to be described.
100		6	Delete *is* so that the sentence reads "the reader will be referred repeatedly to . . ."
101		35–37	In this sentence, *evidence* should be replaced with *confidence*.
103		27	The correct spelling of the author's name is *Kheifets*.
206		7–11	This statement should be changed to read "exposure from VDT work varies from very weak to negligible due to the historical trend toward lower emission levels; however, exposure accumulated over a period of extended use during the day may represent a substantial proportion of daily exposure." (see Mezei et al. Household appliance use and residential exposure to 60-Hz magnetic fields. *J Expo Anal Environ Epidemiol* 2001;11:41–49).
206		34	It should be noted that technically, IARC classification is for cancers.
206		49–51	This sentence refers to "the lower quality previous studies"; I believe the Bracken study is not of lower quality.
242		33–36	A "probable human hazard" classification is not usually granted based on "limited" epidemiological evidence.
272	17.2.15	Row 1, column 3	The entry for the causal hypothesis is missing here and in all similar tables. I do not see the logic as can be viewed as a symmetrical to "no-effect hypothesis"..
273		31–36	Diagnostic classification was based on death certificates in the Savitz study. This point should be discussed.

Policy Options Summary

Specific Comments

Page #	Table#	Line # or Comment # in Table	Comments
8		4–6	The potential increase in exposure due to use of "distributed generation" also needs to be taken into account.

Instead of declaring the Risk of leukemia in children, the write up editorializes the supposed Risk by commenting that there is a 50% increase in risk. I assume this means the risk is 1.5, right down there with second hand smoke. Oh Well, It' s a living.

Has there been any Biological Credibility attached to the fiction about Power Lines? HOW does a Power line magnetic field cause anything in humans?

Are you aware that without medical proof in the form of testing that proves the hypothesis, a risk of 300% (not 50%) is REQUIRED before there is considered enough Risk to even mention Risk? I suppose if you can scare enough people by not telling them you are running worst case simulations based on sample opinion polls, and your results are all estimates, you can make a few bucks, but as for me, without the Scientific Method, described in any Dictionary as a basis, it's still the old "Chicken Little" gag. Maybe someday the public will catch on and start demanding proof before throwing money away on computer games played by doctors.

Charles I. Klivans, cklivans@jps.net <mailto:cklivans@jps.net>

Ray—

Here are my comments on the EMF report. Please forgive the narrative form, but it is one in which I am more comfortable.

First, I found the report complete and thorough in its review of the major studies on EMF and disease endpoints. This completeness is both its strength and weakness. I wanted more emphasis on the meta-analysis that is embedded in the report and less on the Three Wise Men who were your commentators. It is clear that one of the commentators is "more liberal" in his judgment about what risks mean and how much data one accepts by inference.

Second, the most dramatic finding—the linkage with miscarriage rate—is the one most likely confounded by other variables. Miscarriage is the most sensitive marker of reproductive toxins generally. Solvent exposures, and to a lesser extent, solvent-contaminated drinking water, appear to promote miscarriages before there is evidence of any other variable, with the possible exception of low birth weight and/or pre-maturity.
For instance, the most recent British Medical Journal (18 Aug 01) features a fast-track article on effects of proximity to landfills that shows low (or very low) birth weight is the most consistent linkage followed by "all anomalies combined". Most interesting, the observed RR's are all extremely close to 1.0, but nonetheless significant because of the tight confidence (99%) intervals. The commentator for this article points out that even a minor presence of a confounder, say 10% above the norm, that increases the risk by 2X could shift the relative risk by 5-9% (See McNamee and Dolk, BMJ 323: 351-2, 2001).

In the instance of EMF fields, it is entirely plausible that EMF exposure is a consistent surrogate for one or more other factors, e.g., SES-related toxic exposure or just SES itself) which correlate with increased morbidity for the developing fetus.

The "above the line" RR's (which even an amateur like me can see) are so consistent with this and the two other major findings (childhood leukemia and all cancers—cf Floderus's Table 12.2.1 on p 173) that **something** related to EMF is clearly weakly associated with increased risk and those endpoints. I would rather see time spent exploring confounding, looking for dosage effects (as you summarized as I recall some 2 years back in the Amer J Epi review on EMF fields), and most interestingly, any "before" and "after" EMF exposure studies than is now spent on parsing out probabilities.

Third, you note that the adult brain tumor work (coupled with the leukemia linkage) which shows "good" evidence of linkage can be used to reinforce the childhood brain tumor work (see page 144). However, you may be dealing with two different tumor causing events here, which may explain why the evidence for childhood tumors is weak (See the distribution of tumor types, children vs adult). Considering how much rarer childhood brain tumors are than are those in the adult, this possibility should be given credence. However, unlike the adult brain tumors, which have a highly consistent pattern of relative risks above 1 across studies, the children studies are weak (in terms of power) and fluctuate above and below an RR of 1.0 with enough regularity to make the association questionable. Don't tarnish a good correlation (adult brain cancer) with a poor one (childhood brain cancer) just because they sound alike.

Fourth, I would concentrate on the big picture result of your findings: that for all but miscarriages, the slight incremental risk to cancer or disease end-points (ALS) even if real are relatively trivial. Hence, you properly highlight miscarriage risk in your Statement for the General Public, then immediately defuse and evaporate what might otherwise be a (proper) cautionary note by saying most women exposed to EMF do not miscarry. Most women exposed to thalidomide had no birth defects, and most persons exposed to sub-lethal radiation (after Hiroshima and Nagasaki) did not develop cancer. But we take steps to prevent recurrences of these frightful, risk-laden exposures.

If your findings on miscarriage studies are reinforced by careful analysis and detailed scrutiny for biasing factors, then I think it highly appropriate for you to say how much risk appears to be associated with exposure. To reach the point of being willing to put such a statement in your Public disclosure, spend more time in the body of the report ruling in or out the miscarriage idea—if true, you would also expect to see post-partum evidence of damage, e.g., low birth weight, or very low birth weight, etc. If the increased miscarriage rate is even only "largely attributable" to EMF you have an explosive finding of your study. This linkage is so much stronger than the cancer ones, as to leap off the page: but it is also the most difficult to pin down because of the high background "noise" of miscarriages (some studies show more than the 10-15% you acknowledge). I would recommend your speaking with Brenda Eskanazi about this—perhaps a small seminar sized colloquy would be useful.

If you go with the "flag the miscarriage" route, it is probably important to stress that in-home appliance exposure and wiring may give the highest force fields of importance for pregnant women, as you start to do on page 2. . We (working with the March of Dimes) find the evidence on miscarriage to be sufficiently convincing for use to act on the Precautionary Principle and recommend that MoD alert women to the possibility, however hypothetical, of risk to avoid exposures during pregnancy—but where in your report do you help us (or the women) out by giving customary force field data on different appliances—it should be an Appendix.

Fifth, you are right that even while the relative risks are so small to only cause incremental increases in cancer risk, many of these increases pass the regulatory threshold of 1 in 100,000 and hence deserve some kind of notice. But unlike point source pollutants, or environmentally mediated exposures (via air, water, etc), EMF is pervasive, undetectable (by normal means), and constitutes an ever shifting, fluctuating force field that is virtually impossible to control, much less regulate.

Nonetheless, this amorphous EMF "bogey man" creates isopleths of risk that some persons, notably pregnant women, should be aware of: could you not identify the "hot" EMF generators (e.g., Waring blenders? vacuum cleaners? etc) in your report and recommend their avoidance? (Or is that a job for us policy implementers?) Or do you feel even under the Precautionary Principle, those types of warnings generate rather than diffuse anxiety?

Sixth, I do not like or find useful the "**10-50% possible**" language in your executive summary. You mix the "possible" language with subjective statements of the size of the risk in a confusing way. I would rather see terms that specify what risk, according to whom and why: e.g., replace the *"It is "more than 50% possible" that EMF's at home or at work could cause a very small increased lifetime risk of childhood leukemia…*" with
"Our review indicates that it is probable that EMF's at home or at work could increase the lifetime risk of childhood leukemia, etc" and then say by how much, why you think this is noteworthy, and to what extent consumers should be concerned (if at all) as compared to policy makers. It is completely disconcerting to read the sentence as now written, then to find your statement at the end, which says in effect "or it means nothing at all". What is the message???

I don't think the final can use the in quotes possibility estimates: they should indeed be translated into simple English: if something is more than 50% possible, it is "**probable**". If something is only 10-50% possible, it may be "unlikely" (when at 10-30%) but "**possible**" when 31-50%, etc. A simple glossary that gives these interpretative figures would help the reader more than seeing the full-blown and awkward language.

Also, you might want to ask your consultants to agree upon and use simple probabilistic language established by behavioral scientists for instance in genetic counseling: most persons interpret a 1 in 100 risk to be low, but a 1 in 10 chance to be high—often depending on the outcome being considered. Thus, your saying "EMF's at home or work cause a very small increased lifetime risk of leukemia" (p. 1) should be re-translated into more straightforward language: here again a glossary table would help. (See the work of Sorenson and his colleagues at Princeton). Or you might put the whole idea of likelihood of the risk occurring and the strength of the association into a single phrase: e.g., "The likelihood of leukemia occurring in a child exposed to electromagnetic fields over his or her lifetime is real but small: (then specify both the probabilities). etc

I would consider the risk of a 40% increase in miscarriage rate to be outrageously high; but the 5-10% increase high, but perhaps tolerable. (Some persons who have experienced miscarriage would be reassured that they have a 90% chance of getting a pregnancy started, assuming that the risk ended there).

If you wish, I could offer some language here: the tight rope you walk is that you must avoid making the value judgments about how the risk is to be interpreted by the public, while neither underplaying nor overplaying what the numbers mean to a "person on the street" versus what they mean to an epidemiologist.

I find your end of the line with its "don't worry be happy" (viz. "There is a chance that EMF's have no effect at all") to be similar to the "relative risk" comparison sheets put out be industry ("your risk of getting cancer from a teaspoonful of peanut butter are sixty times higher than drinking a glass of water with 20 ppb of trichloroethylene in it") to be ethically distressing and dangerously misleading: You are supposed to be protected from the aflatoxin B contamination of peanut butter by regulatory agencies that do in fact keep contamination levels at or below an appropriate "action level"; you consent to this risk inferentially in all food consumption scenarios: however, you did not consent to being exposed to the carcinogenic risk posed by TCE, nor are there any tradeoffs in drinking contaminated water as compared to foods (peanut butter is nutritious, contains anti-oxidants which reduce risk, etc).

If you consistently see an elevated risk (e.g., 17 out of 19 relative risks in that many studies going above 1.0) and you've done the meta-analysis to show that the likelihood of those risk numbers being elevated is highly unlikely to be due to chance (e.g., where the 95% or 99% confidence intervals are all above 1.0), then you have every right to strike that last part of the sentence, or at least to modify it to give your opinion of why you believe that the risk is not one where "EMF's have no effect at all".

You can't be so ambivalent when there aren't two sides to the story (or at least a reasonable possibility of equipoise) for some risks, e.g., miscarriages and adult brain cancer from EMF while there are for others, e.g., childhood brain cancer from EMF).

The final paragraph which points out the 95-99% of highly exposed persons wouldn't get any of the adverse endpoints is not a good way to help the public understand what an incremental risk is: it is also dangerously poor English construction to begin the phrase "With the exception of miscarriage" since this invites the interpretation that the increased risk of miscarriage does indeed affect perhaps 95-99% of the public.

Finally, instead of petering off with "There are ways to avoid these uncommon accumulated exposures", why not say that **"Because of the likelihood that some of the risks we've studied and observed in highly exposed populations, we believe it appropriate to take preventive steps to reduce or minimize exposure to electromagnetic fields. Even in the absence of complete data and with the residual uncertainties about the size of some of the risks we've looked at, we nonetheless urge precaution in exposure of particularly vulnerable populations, especially children and pregnant mothers to strong EMFs. The most likely sources of these exposures are:…."**

Ray—you've done a splendid, even Herculean job in pulling all this together. Don't dilute the message by being caught up in the probabilistic jargon of the risk assessor: and call the spades as spades where the risk is likely to prove real, affect people early in life, and/or produce humanly unacceptable harm or damage.

Ray--

Your partitioning of the ethics community into the social justice/utilitarian camps peels back only a small part of the cover as I'm sure you know. From my perspective, the key questions about risk include the end point at issue. Greater concern may be more properly focused on early damage in life that produces lasting effects, (e.g., if birth defects were linked with exposure) than on later damage that only chips off the end of life (e.g, a late appearing brain tumor, or even ALS).

Paradoxically, the degree of suffering associated with exposure consequences may be infinitely greater from ALS than from a miscarriage event (certainly for the lost embryo). Hence, it is worth devising a scaling measure for severity of consequences in any ethics based risk assessment.
The old dodge of using QUALS (quality life years) lost leaves me a little cold here, but some index of degree of harm is useful.

Secondly, the "who benefits, who gets hurt" issue is important ethically. If the use of electricity is disproportionate to the risk, e.g., if power grids carrying electricity were centered over low-use, poor income neighborhoods, the moral duty to protect the at-risk population is greater than if there is parity in risk and benefit.

Third, the issue of disclosure is important here. The present way in which your report constantly downgrades risk is ethically inappropriate and disturbing (at least to me). Your statement that "anxiety itself has health consequences" is true, but can lead to unacceptable paternalistic statements.

Fourth, you have an issue of the intrinsic uncertainty of risk, aggravated by the absence of compelling evidence of the residues of harm, for instance (Where are the DNA adducts from emf??). What then do you do in the absence of "clean, certain, cold facts"? For one thing, say that what you have are suspicions, concerns or general issues with unprotected exposures. You can also state what risks can be abated by personal action. For risks that cannot be so abated, you have the issue of exposure in public places: certainly, school environments should be screened for emf field intensity and abatement measures taken, e.g., to bring them within 2mG. The degree of certitude needed to do this may properly be prepared with the tremendous expenditure to reduce the consequence of another low-probability event, earthquakes. If retrofitting of schools for earthquake safety is done with public dollars, so might retrofitting to reduce emf risk, all other things being equal. (The cost-benefit analysis you made, based on a $5M per life estimate is pretty bogus when it is applied equally to leukemia/brain cancer risks in children vs adult onset risks such as brain cancer or ALS).

Fifth, the issue of non-consentual exposure is both a lightning rod for activist reaction, and a bona fide ethical issue. While we are all passively exposed to risk, "cosmic rays" come to mind, we do not all take them with equal equanity. If some emf are intrinsic to living in a technological society, those risks could be characterized as "risks of the commons". The key here is to properly quantify the actual degree of risk. If emf common risks are relatively uniformly distributed, unavoidalbe and commensurate with the gains (say from electrification), than a public relations campaign directed to disclosure, demystification and acceptance is in order. This assumes the risks from emf are comparable to other "unavoidable" risks.

Sixth, there are clearly circumstances where some persons are at unacceptably high risk, be it from faulty wiring, high intensity power lines, or in-home appliances. Only the latter are within the ambit of personal control. Hence, the public health obligation to extend a safety net over the at-risk population--say those within 500 feet of high intensity lines, is strong.

Seventh, the present Policy report is top heavy on the utilitarian calculus and light on the ethical analysis. The social justice issues are not well developed, and are limited to using traditional notions of at-risk groups, e.g., racial

minorities, etc. Your statement about the "precautionary principle" on the top of page 2 is a good start, but has much more richness than the single case situation you identify.

I commend you on even putting a tail on the larger dog that analyzes the study--it was highly responsible not merely to loft the powder keg findings of your large report, unadorned with any policy review, into the public arena. But I do not believe what was produced, while necessary, was sufficient.

QED: you need a bigger report on the Policy Options that flow from your important summary of the science. The tail end of the overall report (the second blue book) is a good start--but is incommensurate with the amount of thought and energy you put into the big blue report which gave the meta-analysis of the science. You could surely use a more robust ethical/policy assessment. (Why not contract with a group like ours to do it???).

I hope these comments prove useful.

Best,

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COMMENTS ON THE EVALUATION ON THE POSSIBLE RISK FROM EMFs BY THE CALIFORNIA EMF PROGRAM

by Patrick Levallois md
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ANSWERS TO YOUR QUESTIONS

1. Yes. It is possible that part of physics is not enough developed to be able to explain possible effects of EMF in humans
2. Yes, they seem really reasonable
3. No, there has been a lot of mechanistic studies and their mostly negative findings should influence more the confidence on the causality of the epidemiology findings
4. No, the mostly negative findings of the animal studies are a strong argument against causality. But I agree that this cannot dismiss totally the epidemiological results
5. Yes, a RR of 1.5 can have a very important population impact. Unless evidence of bias or confounding, it should be considered seriously.
6. Yes and No. Yes in general. But not really when you consider some specific illness as adult leukemia. As those sub-types of leukemia are in fact several kind of disease, environmental agent may act more specifically on a subtype of leukemia and not on all kind. I don't see how the observation of non specificity may increase the degree of confidence
7. Yes. This was done as it has never been before in all kind of experts group working on this kind of issue
8. 50-90% = highly possible, 10-49 % slightly possible

SPECIFIC COMMENTS

Page 3, line 14: I don't see why adult brain cancer is not in the previous sentence
Page 7, line 21: it should be good to specify is that when you consider the cut-off of 3 or 4 mG
Page 9, lines 44-45: it would be good to have more details on how this has changed the result of the evaluation of the 3 reviewers
Line 64-65: IARC classification is not completely driven by the animal studies
Page 14: line 2: again adult brain cancer should move to possible human carcinogen
lines 10-50: too long in a summary. The Disparities section should be summarized here.
Page 17, line 1: I don't see how the 4 % could be applied to various conditions listed in table 2 without more details about its validity.
Table 2: title not clear
Line 22-23: I don't see how we could ajust the PAR by the degree of confidence. This was not the purpose of the degree of confidence.
Page 18, line 10-11: again false reasoning.
Page 20, line 8: give the Webb address
Page 31, line 34-35: Neutra 2001 is not found in the reference section
Table 3.1.1: References are lacking for several exposure metrics
Table 3.1.2: precise the units of measurements (mG?)
Page 33, line 15: Zaffanella study is referred to 1998 in the reference section.
Table 4.1.1: precise somewhere in the bottom of the table that theses arguments were extracted from the NIEHS report

Page 39, first sentence: you should give more details on the studies (and how they were chosen) that were reviewed by the California reviewers and those who were not. It is not clear what you have added to the NIEHS report
Page 64, lines 6-36: this summary on the embryo results is too long
Page 73 : Figure 8.1.1 and 8.1.2 (and it is truth for all your figures of results): give details on what was the exposure metric and cut-off considered in those studies. Delete the two 00 after the comma when you list the studies (number 1 and not 1.00), same thing in Table 8.1.1
Page 88, Arguments for and again causality: I don't understand why you present and discuss the childhood and adult leukemia in the same time. They disserve specific discussions
Page 100: The presentation of assessment by each reviewer is interesting but too long and boring. I recommend to present here only the conclusion and to put the details in an annex
Page 298, line 20, it is a limit (not to adjust for confounders). You should give more details on the possible bias (or not) not to have considered them.
Page 319: Bibliography. Be careful, there are a lot of inconsistencies in the references as they are presented. Some are lacking (ex:Neutra 2001), a lot are incomplete (ex: DelPizzo 1997 or NIEHS 1999), a lot are written as in press but are now published and you should put the good reference. You also forget several times to underline the title of the Journal (ex: Zaffanella 1998).

EDISON ELECTRIC INSTITUTE

701 Pennsylvania Avenue, NW
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Via E-Mail (Jcollins@dhs.ca.gov)

September 10, 2001

Mr. Jack Collins
Program Administrator
California EMF Program
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Oakland, California 94612

Re: Comments on: "An Evaluation Of The Possible Risks From Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations And Appliances" and "Policy Options In The Face Of Possible Risk From Power Frequency Electric And Magnetic Fields (EMF)"

Dear Mr. Collins:

Attached are the comments of the Edison Electric Institute (EEI) on the two California EMF Program reports referenced above. The comments were prepared for EEI by the Exponent Health Group. EEI is the association of U.S. shareholder-owned electric companies, international affiliates and industry associates worldwide. Our U.S. members serve over 95 percent of all customers served by the shareholder-owned segment of the industry. They generate approximately 70 percent of all the electricity generated by electric companies in the country and service about 70 percent of all ultimate customers in the nation. EEI's mission focuses on advocating public policy; expanding market opportunities; and providing strategic business information.

The health and safety of the general public, our customers and our employees are of prime importance to the members of the Edison Electric Institute. Due to the importance of reliable and economical electric power to the health and well being of our society, it is important to reach a socially responsible resolution of health issues regarding power frequency EMF. It is in this spirit that EEI submits the attached criticisms of the two California EMF Program reports.

Sincerely,

Richard M. Loughery
Director, Environmental Activities
and EMF Issue Manager

Attachment

Exponent™

Commentary on:

An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances

And

Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields (EMF)

Two Draft Reports from the California EMF Program

Prepared by the Exponent Health Group for the Edison Electric Institute

Introduction

The EMF Program at the California Department of Health has released two reports for public comment. The first summarizes an evaluation of possible risks from EMF while the second describes alternative policy options for addressing potential risks of EMF.

The following comments are aimed at raising major issues that should be addressed in the preparation of final versions of these reports.

Commentary on:

An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances

Limitations in the application of the decision analysis model

The main method chosen to summarize the reviewers' evaluation of the EMF literature was their rating as to "their degree of confidence that a problem might exist" for 13 different diseases or conditions. The problem is that the confidence ratings were obscure as well as not always based on a detailed evaluation of the primary studies. Therefore, the confidence ratings provided by the reviewers are more superficial than the procedures of a sophisticated Bayesian methodology would suggest. A thorough review indicates that the reviewers achieved no greater insight into the evidence for and against the possibility of health risks than might be obtained by simply asking these three, or any other scientists, for their opinions.

The use of the methodology by such a small sample of scientists whose expertise is almost exclusively limited to epidemiology provides incomplete information. Its application was furthermore so crude that at best it could discriminate the reviewers' evaluation into three categories, **very unlikely**, **possible (to varying degrees)**, and **highly probable** that EMF is a cause of disease. The results of the tallying of confidence scores were that the reviewers classified risks of "birth defects, low birth weight, neonatal deaths, or cancer generally" as "very unlikely". The reviewers rated *no* health condition as "highly probable" of being caused by EMF. All other diseases and health conditions were rated "10-50% possible" or "more than 50% possible," neither of which is clear or intuitive. Thus, the potentially useful 'bottom line' of the confidence ratings is

- that it is improbable that EMFs are a cause of any specific health risks,
- nonetheless, if there are any risks at all, the reviewers have some degree of confidence that EMFs might possibly have causal links to some specific diseases, and

- Cancers in general or neonatal toxicity are unlikely to be causally linked to EMF.

A questionable method was used to assess the consistency of the results of epidemiology studies

The reviewers consider that the data suggest causality if the number of studies with relative risks above 1.0 is greater than would be predicted by chance. Based on this they conclude that chance is an unlikely explanation for the "consistent positive associations" in the literature.

The use of this method is questionable on both epidemiologic and statistical grounds. Analyses of epidemiologic data should consider the quality and power of the study, the nature and relevance of the exposure assessment, and differences among studies, including possible sources of bias and confounding. The method used by the EMF Program reviewers to assess consistency ignores all sources of bias, including obvious information or selection bias, measurement bias or deficiencies in the measurement protocols.

Of particular importance, is that their method also fails to capture the inconsistencies in the estimated relative risks that may be reported within a single study. It is common for EMF epidemiology studies to report relative risks <1, around 1, and > 1 for different aspects of magnetic field exposure within a single study. This leads to the question of how the reviewers selected some risk estimates but not others to describe the study findings.

From a statistical perspective this 'voting counting' method is also inadequate because it accords studies with small or large risk estimates or small or large sample sizes equal weight. Such characteristics are important in the assessment of individual studies and should not be glossed over.

The EMF Program evaluation minimizes the importance of relevant whole animal bioassays yet gives considerable weight to irrelevant experimental studies

In the absence of unequivocal results from epidemiologic studies, as is the case with EMF, experimental evidence needs to be considered in reaching a balanced assessment of potential health risks. In recognition of this need, the National Institute of Environmental Health Sciences (NIEHS) and the Department of Energy (DOE) initiated mechanistic, cellular, and laboratory studies under the EMF Research and Public Information Dissemination (RAPID) Program in 1992 "to clarify existing associations identified from population studies" (NIEHS, 1999).

The RAPID Program was conducted over six years, and at a cost of approximately $46 million was the largest concentrated research effort to date. However, the EMF Program reviewers seem to have dismissed the contributions to scientific knowledge obtained from this program for no valid reason. They noted that their confidence ratings were hardly affected by the results of biophysical studies and analyses and they accorded little evidentiary weight to the results of whole animal bioassays.

Chronic bioassay studies, such as those supported by the RAPID Program, are widely used by scientists and by health agencies to assess potential health risks to humans of chemical and physical agents. The virtual dismissal of experimental evidence by the EMF Program is inconsistent with accepted risk evaluation methods.

In contrast to the lack of weight accorded *in vivo* studies, the EMF Program appears to accept with out question the value and relevance of *in vitro* studies and use their findings in support of hypotheses about EMF effects on wholly unrelated health endpoints. The weighting of *in vitro* data over *in vivo* data is opposite to the reliability that the EPA assigns to these two kinds of studies (e.g., EPA, 1999). Furthermore, the EMF Program provides no basis whatsoever for accepting studies asserting random biological effects, with unknown relevance to any disease, as having direct relevance to cancer or other health effects.

The evaluation of potential miscarriage risks by the EMF Program suggests 'favoritism' towards their own research

EMF Program review of the data regarding miscarriage overemphasizes positive associations in epidemiology studies that are likely to be a result of chance or bias; weighs the laboratory evidence in a manner inconsistent with standard practices in evaluating research; and places undue weight on as yet unpublished studies that the reviewers themselves have either conducted or supported.

For example, the reviewers have concluded that the results of epidemiology studies on computer monitors (VDTs) and electric blankets show an association with miscarriage. Yet they consistently minimize both the role of chance or bias in those studies, and the limitations of the individual epidemiology studies. They are alone among review panels in concluding that the epidemiologic data suggest that EMF from VDTs plays a role in miscarriage (e.g., NRPB, 1994). In spite of the general absence of an association between the use of electric blankets and miscarriage, particularly in prospective studies that have estimated magnetic field exposures from measurements (Belanger et al, 1998; Bracken et al 1995; Lee et al, 2000), the reviewers still lean towards a causal interpretation.

The reviewers' opinion that miscarriage and EMF are possibly causally linked arose from two studies from the EMF Program that included personal measurements (Lee et al, 2000, Li et al, 2001). These studies have yet to be published in peer-reviewed journals but are included as appendices to the risk evaluation report. The EMF Program ignores standard scientific practice by relying on one of these studies prior to feed back from the peer review process of a scientific journal, and accepts the findings of these studies without question despite serious limitations posed by potential response bias over those reported by another larger prospective cohort study without such flaws (Bracken et al, 1995). Without good justification to be found in the risk evaluation, one has to consider the possibility that the reviewer's strong reliance on these studies reflects favoritism towards studies that they funded or led. In addition to their slanted review of the epidemiology studies of miscarriage, the reviewers minimize the significance of large and

well conducted mammalian studies that suggest the absence of adverse effects of EMF on reproduction, mistakenly interpret the results of chick embryo studies as being relevant to the estimation of miscarriage risks in humans (Brent, 1999; US EPA, 1991; 1996), and judge exposures to highly artificial pulsed magnetic fields as more relevant for health risk assessment that exposures to common power frequency magnetic field exposures with largely sinusoidal waveforms.

Commentary on:

Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields (EMF)

As a public health agency the California Department of Health Services (CDHS) has the responsibility for evaluating risks to the California public and developing policies that are protective of health. At this time the EMF Program has requested comments and reviews of its risk assessment report but has not proposed any policies or actions. As a basis for discussion the EMF Program reviewers describe four different policy frameworks: **utilitarian**; **social justice**; **non-interventionist**; and **virtual-certainty-required** that might be considered in the face of uncertainty about health risks of EMF. Unfortunately, these policy frameworks represent mutually exclusive abstract conceptualizations and none correspond to a reasonable blended perspective that should represent the position of the CDHS. In short, the CDHS has provided no guidance, and has simply substituted process for decision-making.

Instead of presenting a public health perspective that addresses utilitarian and social justice goals while making policy based on the best available science, the EMF Program policy option analyses are focused exclusively on the utilitarian policy process. The analyses estimate the costs of mitigating TWA exposures > 2 mG in residences from transmission, distribution, and grounding sources.[1] The costs of mitigating TWA

[1] Ironically, the reviewers' confidence in a causal relationship between EMF and health effects is buoyed more by appeals to unknown 'ingredients' in a magnetic field 'mixture' than by reported associations with TWA exposures, which are the basis for the mitigation plans.

exposures > 2 mG in schools are also estimated and include another source, electrical panels.

Among the problems and limitations of the EMF Program approach to presenting policy options are:

- The only policy option given serious consideration is mitigation; none of the policy projects dealt with any other policy options that have been considered by other agencies such as 'prudent avoidance' of fields by individuals, education, communication, etc.

- The EMF Program reviewers argue that "There is no technical resolution to these kinds of arguments [choices between the policy options offered]. A democracy handles them through the political process." However, the EMF Program reviewers have presented these options as extremes and as mutually exclusive and have not offered public health options that are appropriate to the uncertainties in the risk assessment and the extremely low estimated individual risks. The EMF Program reviewers approach is also inconsistent with past CDHS actions and those taken by other state and federal health agencies. If applied generally, the EMF Program approach would politicize public health policy and substitute endless battles between abstract, unsupported policy option frameworks, for action.

- The EMF Program evaluates the speculative impacts of EMF in isolation, which is contrary to a balanced public health approach. The EMF Program should have considered whether an estimated expenditure of $480.5 million ('modest cost measures') or $7.61 billion ('expensive measures') were appropriate in relation to state expenditures for other public health issues that are based on clear and convincing science (e.g. prevention of the spread of West Nile virus, education for AIDS prevention, etc.). The California Office of Environmental Health Hazard Assessment has established a comparative risk evaluation framework based on the recognition "that focusing on environmental threats in isolation, rather than collectively, had resulted in a misdirection of scarce funds to less serious environmental problems" (OEHHA, 1994, p. i). Such comparisons would reveal the inappropriateness of their recommendations for EMF mitigation.

- The EMF Program reviewers argue "The PUC is unlikely to authorize the investor owned utilities to spend rate payer money on smoker education [a potentially even more cost beneficial use to which scarce resources could be put], so that question is not realistic." This is a specious argument. The issue is not ratepayer dollars, but society's expenditures, since all society ultimately bears these costs. If the science actually justifies the implementation of public health measures regarding EMF, then the EMF Program should recommend action to CDHS regardless of the potential source of funding. The CDHS is responsible for providing guidance to the state on all health issues and establishing priorities and

plans for protecting public health. The funding to support that guidance is the responsibility of the legislature, not just the PUC.

- No health agency has concluded that associations of EMF with disease, with the possible exception of childhood leukemia, are likely to be causal in nature, even though the possibility has not been excluded. The EMF Program reviewers have not provided any persuasive evidence for a relationship of EMF to these other diseases and the inclusion of other health endpoints besides childhood leukemia is taken to justify far reaching and expensive EMF mitigation measures. The EMF Program reviewers have argued that "expensive measures would not be justified by even a 100% degree of confidence of a quite strong effect on this disease [childhood leukemia] alone." and this apparent need to justify mitigation may explain why their evaluation appears to torture risks from the data for conditions besides childhood leukemia that cannot be justified at face value.

- Despite the many years of effort and large expenditures of the EMF Program run by the reviewers, they provide no clear rational response to residual uncertainty about the possibility of EMF risks. There is no certainty that the expenditure of vast sums on mitigation efforts will yield any public health benefit.[2] Other actions including providing public information and targeting research to reduce uncertainty are more appropriate, given the current status of research. These aspects of the EMF Program should be continued and not be replaced with a permanent regulatory structure that would be ill suited to serving public health needs.

In contrast, on the basis of their assessment of EMF research including the RAPID Program, the NIEHS recommended no aggressive regulatory action (NIEHS 1999).

Conclusion

In broad outline, the evaluations of leukemia offered by the EMF Program reviewers do not differ widely from those published by other organizations including IARC, NRPB, and NIEHS. However, their assessment of ALS differs, and their evaluation of EMF as a cause of miscarriage is clearly stronger than the conclusions of other reviewers, both on the likelihood and on the magnitude of risk. For miscarriage, they have estimated large, not small, potential risks to the population. In comparison to other scientific organizations, the EMF Program reviewers point to a potentially wider spectrum of health outcomes for additional investigation. What must not be lost in the discussion of

[2] All of the reviewers' confidence ratings that EMF is a cause of 13 diseases or conditions are "possible" or "unlikely" Both ratings imply that "there is a chance that EMFs have no effect at al." (p. 1).

process, opinions, and percentages, is that the evaluation still includes the strong possibility that there is no risk at all.

The confidence that scientists and public health officials can place in the EMF Program judgements is seriously undermined by the problems in the clarity of these reports and their conclusions, in the conduct of the risk evaluation and in the failure of the EMF Program to advance public policy appropriate to the weight of the scientific evidence. Therefore, the evaluation and methods of the draft reports should be revised and redone by a review team wholly independent of the EMF program.

In spite of the deficiencies in the EMF Program reports, attention should not be diverted from the need to "continue targeted research into EMF and health, exposure assessment, and field management as recommended in the RAPID Program's "NIEHS Report on Health Effects from Exposure to Power-Line Frequency Electric and Magnetic Fields" (EEI Policy Position on Power Frequency Electric and Magnetic Fields (EMF), January 11, 2001). Research of high quality to address gaps and uncertainties in existing scientific knowledge will provide a stronger basis to assess the need, if any, for an updated public policy on EMF.

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September 8, 2001

Dear Mr. Collins:

Enclosed is a document that constitutes my comments on the Risk Evaluation Report from the California EMF Program, and also include some comments on the Policy Options Summary.

Very briefly, I point out that there are currents of other frequencies besides 60 Hz present on today's electric power lines, and these have been completely disregarded; that is, the health effects of the fields from these currents, which have frequencies higher than 60 Hz, have not been evaluated to date; and the possibility that some of *them* may be causing the diseases that have been associated with proximity to electric power lines and with certain strengths of 60-Hz magnetic fields around electric power lines has not been considered by the experts relied upon in the report of the California EMF program.

I also point out that the primary reason that investigators have concluded that 60-Hz fields are hazardous to health is that there is a recurrent pattern that associates certain diseases with certain values of 60-Hz magnetic field strength. The experts cited in the report from the California EMF program have decided that this association is *not* a chance occurrence (with which judgment I concur) and therefore the 60-Hz magnetic field is the agent causing these diseases (an unjustified leap to an incorrect conclusion, in my judgment), and the strength of the magnetic field measures the disease risk of exposure to the field (also an incorrect conclusion, in my judgment).

In summary, there is indeed a real hazard to health from the electromagnetic fields that surround electric power lines, but it is not *caused* by the 60-Hz magnetic fields present around these lines. Instead, the observed health hazard is caused by certain other electromagnetic fields of much higher frequency which are also present around electric power lines-because small currents of such frequencies are present on electric power lines-but which have never been measured and have always been ignored by researchers.

One reason these fields of higher frequency have been ignored is that the currents that give rise to them are much smaller than the 60 Hz current, so that the strength of the 60-Hz field is much greater than the strength of a comparable field at any higher frequency. Researchers believe wrongly, in my judgment-that the strength of a field measures the health risk it poses, and so attention can be confined to the strongest fields, as these should be the most hazardous.

This logic is sound, but its premise is false, so the conclusion is false, also. Interestingly, in the matter of chemical agents, it is widely accepted that this conclusion is incorrect (if we substitute 6 4 concentration" for "field strength"). For example, if a tiny amount of botulin toxin is mixed in a large amount of water and is fed to an experimental animal, which then dies, the scientist does not leap to the conclusion that water-which constitutes almost I 00% of the fluid drunk-is lethal! It is well recognized that constituents present in very small concentration may be highly toxic, and may determine the toxicity of the mixture. It is also recognized the toxicity of a combination pen-nits no inference at all about the toxicity of the constituent that is present in highest concentration, unless this is also the *only* toxic component present.

I submit that the same is true for electromagnetic fields. When there is a combination of fields present, only one of which is capable of injuring health, the hazard from the combination is determined by the hazard posed by the one component that is capable of injuring health.

If more than one hazardous component field is present, the hazard from the combination of fields is determined by some type of "addition" of the hazards posed by the two separate harmful components.

Actually, as theoretical analysis shows, it is *the frequency* that is the best predictor of hazard, not the strength of the field, when other information is absent. The general rule is that high frequencies are more hazardous than low frequencies.

So the 60-Hz fields that the report of the California EMF program labels as hazardous to health are actually quite safe---despite their high strength---because their frequency is so low. What is unsafe, and justly deserving of the label that is wrongly being placed on 60-Hz fields, are some of the long-ignored, unstudied radio-frequency fields that are present around modem electric power lines (and also around the internal electric wiring in buildings, and the electrical wiring in electric appliances such as hair dryers, electric blankets and computers). These occupy a spatial region of limited extent around the current-carrying wire, such as a power line (which is why the *wire code* of Wertheimer and Leeper, which incorporates the distance from the power line as a parameter, is a more effective predictor of disease risk than the measured strength of the 60-Hz magnetic field). The strength of these RF fields is quite small; but they are extremely hazardous to health, even at very small field strengths.

This is the only conclusion that is consistent with all the evidence. After all, if 60-Hz fields were harmful to human health, there would have been evidence of it *long* before the end of the 20th century, inasmuch as electric power has been in use since the late 19th century! But evidence of a hazard associated with electric power lines didn't appear until the last fifth of the 20th century! Any hazard to health from 60-Hz fields such as the report of the California EMF Program's Risk Evaluation Report alleges would have made its appearance much earlier in the twentieth century, were it *truly* caused by exposure to 60-Hz fields!

Also, if 60-Hz fields were *really* the agent of hazard, the replication by David Savitz *et al.* of the initial published study by Wertheimer and Leeper would not have shown that their wire codes correlated better with the health effects than measured values of 60-Hz magnetic field strength did! This quite unexpected result, sometimes called the "wire code paradox", is *completely inconsistent* with the idea that 60-Hz magnetic fields caused the diseases under study, and was the earliest indication that there was a serious error in the hypothesis being evaluated by scientists.

Two paragraphs above, I identify the hazardous frequencies as being radio frequencies. I do this because the health effects associated with electric power lines are radio-frequency health effects. It is not conclusively known just what range of radio frequencies is hazardous, but there does exist a basis for making an approximation, using electromagnetic field theory. I have been trying to do this, but have not yet succeeded in accomplishing it, because of lack of time.

The foregoing is a brief outline of my comments on the Risk Evaluation document, which are presented in detail, with references, in the first part of the enclosed document.

The policy implications, under the scenario I present, are rather different from those discussed in the Policy Options Summary of the California document. I present a discussion of these in the second part of the enclosed document, and briefly summarize them below.

I must mention that there are basically two configurations of electric distribution systems. The delta configuration employs three wires, all elevated above ground potential, none of which is electrically grounded. The wye configuration employs four wires: three "hot" wires at an elevated potential, one for each of the three phases, and a neutral wire at ground potential, which is electrically connected to the earth. A wye-configured system may be electrically grounded at only one point, or at multiple points along its length.

In a distribution system of the multi-grounded wye type, the earth is electrically connected in parallel with the neutral wire, which means that there is an opportunity for electric current that would normally travel on the neutral wire to travel across the earth. The relative electrical resistance of the two parallel-connected paths will determine how

much current travels on each one. The vast majority of electrical distribution systems in the USA are of the multi-grounded wye design at this time. The reason is because this configuration allows a substantial proportion of the current from the distribution system to flow on the earth, if the electrical resistance of the neutral wire is comparatively high. (The electric company saves money, if it does not have to put in place a neutral wire that is much lower in resistance than the earth.)

Whatever frequencies are present on the electric distribution system, including the neutral wire, therefore flow across the earth, in the vicinity of a multi-grounded wye distribution system. If there are harmful radio frequencies present on the neutral wire, these will flow across the earth, making the portion of the earth that carries them a radio-frequency waveguide. I submit that the presence of these harmful radio frequencies on the earth is the cause of the health problems on farms that farmers refer to, collectively, as "stray voltage" problems.

The scenario I present indicates that there is a potential hazard to health not only from exposure to an RF field in air, which is capable of inducing the flow of an RF current of the same frequency in the tissues of an animal, but also from flow of a "contact current" that is not induced by exposure to a field in air, but which is no less hazardous.

In other words, in my comments I identify *an additional health risk not considered in the Risk Evaluation Report of the California EMF Program!*

The major differences between the implications of the scenario painted by the California Risk Evaluation Report and my scenario are as follows:

(1) the health hazard can differ greatly from one electric power line to another, because it depends on the degree of RF "pollution" of the electric power transmitted on that particular power line; therefore, *it is not possible to make generic assertions about the hazard to health posed by all electric power lines.*
(2) the hazards to health currently associated with proximity to electric power lines will vanish for any given power line, if radio-frequency currents in the harmful range of frequencies can be removed from that electric power line ("removed", in this case, means that the current at the frequencies of interest can be made sufficiently small that the hazard to health vanishes, for all practical purposes).
(3) because most distribution lines in the USA are of the multi-grounded wye type, the presence of harmful radio frequencies on electric distribution lines implies that these same frequencies can be present on the earth in certain locations, where they can produce a flow of electric current through an animal by contact (the "contact current" usually travels through the feet, legs and trunk of the animal).

Under my scenario, the way to eliminate the health problems caused by exposure to harmful RF fields in air in the vicinity of electric power lines is to take steps that will ensure the removal of harmful RF currents from electric power lines.

There are two possible ways to eliminate the health problems caused by the flow of harmful RF currents across the earth: to remove harmful RF currents from the electric power system, or to prevent the current that properly ought to flow on the neutral wire of a multi-grounded wye distribution system from flowing across the earth (such as by making sure that the neutral wire of the system is a low-resistance path, in comparison to the earth, for example; or by changing the multi-grounded wye system to a single-grounded wye system or to a delta system).

The strategies that must be developed for removing RF current from electric power lines will depend on the reasons why the RF is present on any given electric power line. There are two possibilities: it was deliberately put there, or it is unintentionally present.

RF current has been deliberately put on electric power lines since the late 1920s for communications purposes. When used in this manner, the RF current is referred to as *power line carrier.*

Most transmission lines in the USA *use power line carrier* to meet their communication needs, and have done so since the 1930s. However, within the past two decades a new, safe communications technology has arisen for this purpose, which uses fiber-optic cable. New transmission lines are being designed to use fiber-optic cable communications systems, but this does not mean *that power line carrier* is disappearing from transmission lines. For example, a proposed transmission line through northwestern Wisconsin (the Arrowhead-Weston line) has been designed to use fiber-optic cable as its primary communication system; but for high operational reliability, it will also have *a power line carrier* system in place operating at reduced power, on stand-by, so that if the fiber-optic cable system should fail, *the power line carrier* system will be available as a back-up system to handle communications until the fiber-optic cable system has been repaired and restored to service.

So the presence of a fiber-optic cable communications system on a transmission line doesn't necessarily imply the absence of *a power line carrier* communication system on that transmission line.

Some distributions systems also *use power line carrier* for such tasks as remote meter reading or remote control of customer loads.

RF currents are also widely present, unintentionally, on building wiring and appliance wiring because there has been a steady deterioration over a period of decades in the *quality of electric power,* due primarily to the increasing use of what electrical engineers call *nonlinear loads on* the electric power system. In other words, what the customer plugs into the electric power outlet can degrade the quality of electric power.

In like manner, the kind of equipment that electric power companies put on the system can also degrade the quality of electric power. The switching of capacitor banks at an electric substation can send high-frequency pulses through the distribution system for quite some distance, for example.

There are two approaches that can be used to bring about the reduction of RF in buildings, on appliances, and on distribution lines. One is to impose a legal requirement on the design of the power supply of equipment (such as computers) that constitutes a major nonlinear load on the system. The European Community already does this, so all that we have to do in this country is to manufacture for the domestic market in the same manner as for the European market. (In Wisconsin, legislation at the state level is being developed in an attempt to accomplish this; the draft version is expected to be available sometime in October, 2001.)

But we still have to deal with the existing installations in homes and businesses all over the USA. A simple, inexpensive way exists to accomplish this; it is being tested in Wisconsin at this time. It consists of installing an RF filter on the electrical wiring of the building, right where the power comes into the building, on the customer's side of the meter. The RF filter consists of appropriately sized capacitors. The filter is simple and comparatively inexpensive; it is installed between the "hot" wire and the neutral wire.

Electric power companies also need to erect RF filters between distribution lines and transmission lines. If RF filters are installed throughout the electric power system in this way, RF can be isolated at its source, and prevented from travelling through the electrical system. (Individual appliances may still be hazardous, but this can be dealt with by manufacturers in the design process and in the meantime, people can take precautions with respect to specific appliances.)

One reason why the situation has become as bad as it is at present is that the state agencies that regulate electric utilities have failed to enforce existing power quality standards. Thus a major change that is needed is for the state agencies that regulate electric utilities to reach a new understanding of what they ought to be doing as part of the process of regulating electric utilities.

This is true with respect to the presence of RF on power lines, and it is also true with respect to the flow of current across the earth, because of the historic practice by the electric utilities of allowing current to flow on the earth that properly ought to have been confined to the distribution system neutral wire.

As power quality is improved and hazardous RF currents are removed from electric wires and electric power lines, the disease risk that makes certain electrical occupations hazardous to health will be reduced, and these occupations will become less hazardous.

In conclusion, let me emphasize that the 60-Hz electric and magnetic fields associated with the electric power transmission and distribution systems of North America are *not* inherently hazardous to health, as the Risk Evaluation Report of the California EMF Program asserts. Many present electrical system wires *are* surrounded by unhealthful electromagnetic fields, just as the Risk Evaluation Report indicates, but *not* for the reasons given. Instead, it is because we have allowed these systems to become "polluted" with hazardous radio frequencies, much as water can be contaminated with toxic pollutants. What needs to be done is to remove the toxic RF "pollution" from our electrical systems. This will restore our electric power to its former high quality, which will then restore the EMFs associated with electrical wires to their original healthfulness.

Yours for a more healthful environment,

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Enc.: Comments on the Risk Evaluation Report [An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFS) From Power Lines, Internal Wiring, Electrical Occupations and Appliances] and the Policy Options Summary [Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields (EMF)] of the California EMF Program, both dated April, 2001

Comments on Draft Documents dated April, 2001, from the California EMF Program

An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFS) From Power Lines, Internal Wiring, Electrical Occupations and Appliances
[in brief, the Risk Evaluation Report]

and

Policy Options in the Face of possible Risk from Power Frequency Electric and Magnetic Fields (EMF)
[in brief, the Policy Options Summary]

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Personal Information

I posses an earned Ph.D. in physics, and am certified in the comprehensive practice of industrial hygiene by the American Board of Industrial Hygiene. I have begun to develop the scientific foundation for a new discipline called *bioelectromagnetic hygiene,* devoted to the prevention of diseases caused by exposure to non-ionizing electromagnetic fields and radiation, and to the flow of such currents through living tissues. My r6sum6 is in Appendix A

Introduction to my Comments

The authors of the various documents that constitute the Risk Evaluation Report conclude---correctly, in my professional judgment---that the association of disease and ill health with power-frequency electric and magnetic fields around electric power lines is a real phenomenon, and is not merely occurring **by** chance. I agree that there is indeed a hazard to health associated with proximity to wires carrying electric current, whether these are electric power lines, or building wiring, or the wiring within an electrical appliance, at this time in history.

Current flowing through a wire sets up a magnetic field around the wire. Our electrical system maintains some wires at an elevated electric potential with respect to the earth, which establishes an electric field in the space around these wires. Therefore the wires of our electrical system are surrounded by both electric and magnetic fields. The authors of the Risk Evaluation Report have concluded that the observed cases of disease and ill health have arisen because of exposure to the fields around these current-carrying wires.

In the main, I concur, though with some differences. For one thing, I prefer to call these fields 6 4 electromagnetic fields" in the general case. At low extremely frequencies, the electric and magnetic fields are separable (as explained in Reference 1), so that it is appropriate to refer to 60-Hz magnetic fields separately from 60-Hz electric fields. But at higher frequencies, the field is more properly referred to as an electromagnetic field. I shall use the term EMF to refer to electric and magnetic fields without any assumption regarding their separability, and without assuming any particular frequency value within the non-ionizing spectrum.

For another thing, the authors of the Risk Evaluation Report have concluded that the agent of hazard is 60-Hz EMF. I disagree strongly because the available evidence is thoroughly *inconsistent* with this conclusion. (This is discussed at length in Part 1, Section I of this document.)

The authors of the Risk Evaluation Report have completely disregarded the presence of other fields (at higher frequencies) around electric wires. They seem to have done so in part because no studies have been carried out on the health effects of such fields, and in part because of an assumption that the most hazardous field is the strongest field present. The 60-Hz fields are indeed much stronger than are the fields of higher frequency around current-carrying wires. Presumably the authors of the Risk Evaluation Report felt justified in disregarding the higher-

frequency EMF because the strength of these fields is quite small in comparison, and for this reason concentrated all their attention on 60-Hz fields. I consider this a scientific error. (Fields of other frequencies that are present around electric power lines are discussed in Part 1, Section 2 of this document.)

The authors of the Risk Evaluation Report seem to have accepted the idea that the strength of an electric or a magnetic field measures the risk to health that exposure to it poses; in other words, that the strength of an electric or magnetic field is an adequate exposure metric for health hazard evaluation purposes. This is true only in a special circumstance, and to a limited degree. In general, it is *not* true; and it most definitely is *not* true of the fields surrounding electric power lines! However, this is a common error made by most scientists doing research in this field, which is probably why the authors of the Risk Evaluation Report accepted it. (What constitutes a good exposure metric is discussed in Part 1, Section 3 of this document.)

While I agree that there is a hazard to health from exposure to the EMF around electric power lines and other electrical wiring, *I emphatically disagree* with the attribution by the authors of the Risk Evaluation Report of this hazard to 60-Hz fields. I consider that the available evidence indicates clearly that fields of much higher frequency-within the radio-frequency (RF) range are the agent of hazard in the vicinity of wires carrying electric current, and are causing the illness and disease that the authors of the Risk Evaluation Report attribute to 60-Hz fields.

The authors of the Risk Evaluation Report have concentrated all their attention on exposure to EMF in air, but (as discussed in Part 1, Sections 4 & 6 of this document) there is an additional hazard to health that comes from the practice whereby electric current from the power system is allowed to flow across the earth; this gives rise to an opportunity for flow of "contact current" through both people and animals. This is a serious hazard to health that is not addressed at all in the Risk Evaluation Report. It also gives rise to some environmental hazards not addressed in the Risk Evaluation Report (which are briefly discussed in Part 1, Section 7 of this document).

Basic facts, including historical facts, about the electrical distribution systems in North America provide a foundation for the discussion of the "contact current" health risk. (These basic facts are presented in Part 1, Sections 4 & 5 of this document.)

Because the Policy Options Summary assumes that 60-Hz fields are hazardous to health, though the available evidence indicates otherwise, I provide some discussion of the policy implications of the scenario I present here, in which 60-Hz fields are innocuous, but the passage of RF current through the body-either induced current resulting from exposure to RF fields in air or "contact current" resulting from the flow of current across a surface-is the cause of disease.

Because there is indeed a genuine hazard to health associated with the EMF around modem electric power lines and electric wiring, some of the contents of the Policy Options Summary document may be applicable to the scenario I describe. But I have not had sufficient time to review the Policy Options Summary document in detail; therefore, I simply discuss in broad terms what 1, as a disease prevention professional, perceive as the tasks that must be addressed in order to reduce or eliminate the hazards to health that I am aware of.

Part I. Comment on the Risk Evaluation Report

Section 1. Summary of the evidence that 60-Hzfields are not a cause of illness or disease

In 1991, the U. S. Congress asked the U. S. National Academy of Sciences to conduct a review of the scientific evidence regarding the hazard to health from exposure to power line electric and magnetic fields in residences. Academy scientists decided that the study should address 60-Hz fields. The final report was released in October, 1996, and the complete report (from the National Research Council) was published as a hardcover book' in 1997.

The Committee on the Possible Effects of Electromagnetic Fields on Biologic Systems, convened by the National Research Council to conduct the study, was asked "to review and evaluate the existing scientific information on the possible effects of exposure to electric and magnetic fields on the incidence of cancer, on reproduction and developmental abnormalities, and on neurobiologic response as reflected in learning and behavior."

Having studied this report in some depth, I consider it to be a sound, unbiased review of the scientific evidence. The basic conclusions reached in this report of interest here are:

(1) "Based on a comprehensive evaluation of published studies relating to the effects of power frequency electric and magnetic fields on cells, tissues, and organisms (including humans), the conclusion of the committee is that the current body of evidence does not show that exposure to these fields presents a human-health hazard." [Reference 1, pages 1-2]

(2) "An association between residential wiring configurations (called wire codes...) and childhood leukemia persists in multiple studies, although the causative factor responsible for that statistical association has not been identified. No evidence links contemporary measurements of magnetic-field levels to childhood leukemia." [Reference 1, page 2]

(3) "Exposure levels of electric fields and other characteristics of magnetic fields (harmonics, transients, spatial, and temporal changes) have received relatively little attention." [Reference 1, page 5]

It is not yet widely known in the western hemisphere, but in Japan a similar evaluation was also taking place, with one difference: the Japanese actually designed and carried out studies, in their evaluative effort, whereas the National Research Council report in the USA simply reviewed already-existing scientific papers.

The work done in Japan was published in the Japanese language, but only recently has a translation into En lish2become available. The conclusion from the Japanese work was in agreement with the first conclusion listed above from the National Research Council report in the USA.

So two separate, independent efforts on opposite sides of the world have been carried out over the past decade, and both have concluded that the available scientific evidence is *inconsistent* with the hypothesis that power frequency (50- or 60-Hz) fields cause human illness or disease.

Let me review, briefly, how the idea that power-frequency fields-initially, magnetic fields might be harmful to human health first arose. It resulted from publication of a paper 3 in 1979 by a budding epidemiologist, Nancy Wertheimer, and a physicist, Ed Leeper. Nancy Wertheimer was doing epidemiological research "on the cheap" as a personal project in Colorado; she had no institutional funding of any kind and was paying for the cost of her research out of her own pocket. As a result, when she noticed an association between residences of Denver, Colorado, cancer eases and proximity to step-down transformers on the electric distribution system, and suspected that the magnetic field around the electric wires might be important, she had no money with which to purchase a gaussmeter that could be used to measure the strength of this magnetic field.

A local physicist, Ed Leeper, helped her out by improvising a simple detector, "using a coil employed by television repairman to demagnetize TV sets and an audio amplifier and speaker from an old walkie-talkie',4 and presented it to her as a Christmas gift at the end of 1974. The output of this detector was an alternating voltage heard on the speaker as a hum that got louder or softer as the amount of current flowing through the coil increased or decreased.

This convenient detector responded ***not*** to the strength of any alternating current magnetic field (the magnitude of the magnetic field vector **B),** but to the strength of its time rate of change (the magnitude of the vector dB/dt) which I

call the "magnetic induction current". Throughout most of 1975 Nancy Wertheimer used this detector to "probe" the field around the electric distribution wires near homes of children in the Denver area who had died of leukemia.

Nancy Wertheimer and Ed Leeper assumed that the signal from this detector was proportional to the magnetic field strength of 60-Hz magnetic fields around the distribution wires, because this would be true, if there were only a 60-Hz field present around these wires. However, if in fact there were fields of higher frequency also present near the distribution wires, then this detector would respond to all the fields present on a frequency-weighted basis, meaning that it could respond more strongly to the high-frequency magnetic fields than to 60-Hz magnetic fields.

When they prepared their paper for publication, they could not present readings from this device, even though they had used it to probe the magnetic field around the distribution wires, because it wasn't calibrated in any meaningful way, and the data from it were not numerically quantified. In order to have something for publication, they developed a *wire code* system based in part on the results from this device, and in part on other parameters such as the size of the distribution line wire and the distance of the residence from the distribution line.

They never presented any actual data measuring 60-Hz magnetic field strength in their paper, but the readers of their paper assumed that, if data on the 60-Hz magnetic field strength *had* been presented, the correlation shown in their paper between the incidence of childhood cancer and the wire code category of the child's residence would have been even stronger; that is, a stronger correlation between the incidence of childhood cancer and the 60-Hz magnetic field strength measured at the residence would have been observed.

Actually, the *initial* reaction to the Wertheimer-Leeper paper was total disbelief. Hardly anyone could accept the idea that 60-Hz fields might be harmful. Electricity had been in use for a century; if the fields around electric wires were harmful to human health, this should have become apparent long ago, shouldn't it?

But questions about a possible human health hazard from the fields around electric power lines continued to arise, so finally a formal epidemiological study designed to replicate the Wertheimer-Leeper study was funded; it was carried out by David Savitz *et al.*[5] In this study, also carried out in the Denver area but using different cancer cases, the actual 60-Hz magnetic field strength was measured inside the residences. The residences in the study were wire coded, too, in the manner that Wertheimer and Leeper had done.

To everyone's enormous surprise, in the Savitz study disease incidence correlated more closely with the wire codes than with the measured 60-Hz magnetic fields! This unexpected result has been termed the "wire code paradox", because it is a well-respected principle of science that if an effect is being correlated with several different possible causes, it should correlate most closely with the *true* cause, and less closely with other parameters that are not directly causally related to that effect.

The result obtained in the Savitz study seemed paradoxical because the scientists were certain that the 60-Hz magnetic field *had* to be the true cause of the observed cancer cases, yet it didn't correlate as closely as did the wire code categories that Wertheimer and Leeper had used as a surrogate for the 60-Hz magnetic field strength!

In summary, the evidence *against* 60-Hz EMF as a cause of human disease and illness includes the following:

(1) evidence from laboratory studies has been consistently negative;
(2) two major national scientific investigations (in Japan and the USA) gave negative results;
(3) a century of human experience with electric power lines failed to produce indications of a hazard to human health; and
(4) the Savitz study produced a paradox, under the assumption that 60-Hz fields cause disease.

Clearly, then, there are some formidable obstacles standing in the way of a conclusion that exposure to 60-Hz EMF causes disease in human beings.

Section 2. Evidence of other frequencies on electric power lines and electric wiring

One properly ought not to conclude that 60-Hz EMF causes disease until all other types of EMF present around electric wires have been ruled as a possible cause of such disease. To date, no direct evaluation of the health effects of exposure to any electromagnetic fields around electric power lines, other than 60-Hz fields, has been attempted.

The logical first step is to identify the other EMF that is present around electric power lines; that is, the fields having a frequency different from power frequencies (50 or 60 Hz). It's well known that there are harmonics present, to some degree, on electric power systems. [A harmonic is an integer multiple of a fundamental frequency. In a 60-Hz power system, the second harmonic has a frequency that is twice 60 Hz; the third harmonic has a frequency that is thrice 60 Hz; *etc.]*

In theory, one might consider an infinite number of harmonics, but as a practical matter, an upper limit is usually imposed. Here in Wisconsin, the Public Service Commission-which is the state agency that regulates electric utilities-pays official attention only to harmonics through the 50th: that is, to harmonics up to 3000 Hz (3 kHz). Frequencies higher than this are disregarded by the Public Service Commission of Wisconsin. (It seems likely that a similar statement is true regarding the state agencies that regulate electric utilities in other states.)

However, current of higher frequency **is** present on electrical systems. An instrument which can be used to measure the RF voltage on building electrical wiring has been described 6 and has been used to make measurements in the state of Wisconsin. This instrument, which consists of an RF meter with a high-pass filter on the input (to take out 60-Hz and low harmonics) is capable of making measurements up to a frequency of 20 kHz.

I have used this instrument to make a spot survey of RF on building wiring across the state of Wisconsin. (These data have not yet been published, although they have been publicly displayed in a poster exhibit. They are presented here in Appendix B.)

Transients-brief voltage pulses-are also present on electric power systems. The frequencies of which these pulses are composed typically lie in the radio-frequency portion of the spectrum. Indeed, transients were identified in the National Research Council report (Reference 1, page 5) as requiring investigation as a possible cause of the diseases that have been associated with electric power lines. (No institutionally-funded research effort to study the health effects resulting from the presence of transients on electric power lines is known to me. I am aware of one privately funded study, the results of which have not been published in the scientific literature.)

There has been a steady deterioration in the quality of electric power over a period of decades. In other words, the amount of current on power lines at frequencies above the fundamental frequency has been increasing over time. This is undoubtedly contributing to the presence of RF current, and transients, on electric power systems, and on building wiring.

But radio-frequency current has also been *deliberately* placed on electric power lines by electric power companies! This is done for communications purposes, and for remote control. The term used in the electrical industry for this use of radio-frequency communication over a wire (sometimes called "wired radio", in contrast to broadcast or "wireless" radio) is *power line carrier.*

In 1997 1 carried out a library study 7 of power line carrier, reviewing the history of its development and use on transmission and distribution lines around the world. Briefly, it was developed in Britain about 1912 for purposes of

remote control and found many applications all over the world. Initially it was used on distribution lines, but by the early 1930s it provided the standard method of communication for transmission systems in the USA, and it has been in widespread use all over the world for this purpose throughout most of the twentieth century. Its use on distribution lines to automate the task of reading electric meters is common in other countries, but less so in the USA.

I provide a few scattered citations to the literature to prove my point. In a 1950 paper, 8 it was reported that in Britain, power lines were being used to transmit the following, at the indicated frequencies:

System	Frequency Range
"D.C." telegraphy	up to 300 Hz
Music channels	30Hz- 10kHz
Voice-frequency telephony	300 Hz - 3kHz
Low-frequency carrier telephony	3kHz- 3OkHz
24-circuit carrier telephony	12 kHz - 108 kHz
Coaxial-cable telephony	60 kHz - 4 MHz
Television	**500** kHz - 4 MHz

I was surprised to read that television has been transmitted over power lines by carrier signals, as I had not known that this had ever been done. I find it interesting that another British engineer 9 stated, concerning carrier transmission over high-voltage lines: "The usable frequency range is about 50-500 kHz, the upper frequency limit being directly attributable to the large spacing between phase conductors (>8 m)." After reading this, I wonder how frequencies as high as 4 MHz could have been transmitted.

In a paper published in 1965, two electrical engineers in Germany reported that they had reviewed the worldwide experience from using power line carrier equipment on 400 kV transmission lines, in order to evaluate its suitability for use on a 735 kV (extra-high-voltage) line. Their conclusion was that power line carrier systems would also provide extremely reliable and economical communications paths for extra-high-voltage power lines. Io

A study' 1 of the propagation characteristics of a 25 kHz carrier signal-including attenuation and velocity of propagation-was done on a 20,847-foot overhead distribution line test site owned by the Carolina Power and Light Company of Raleigh, NC, that was designed for operation at 23 kV. Measurements were made on the line alone, and on the line with distribution transformers whose secondaries were both open and shorted. Conclusions were that the velocity of signal propagation on the unloaded line was approximately 95% of the speed of light in air, which dropped to 85% on the loaded line. The attenuation was well below I dB/mile, even with the line loaded.

The results from this study were important because power distribution lines are often used as a communications medium for load management, meter reading, and other purposes. 12 The signals most often used for this purpose are in the band from 4 to 16 kHz." Notice that these frequencies lie within the "up to 20 kHz" range to which the instrument 6 responds that I used in my spot survey of RF on building wiring in Wisconsin.

Power line carrier radiation from high-voltage lines has been measured 13 and an effort has been made to compare measurements with calculated fields. "The precise calculation of the complete field pattern surrounding a two-wire transmission line above the earth is difficult."14 In addition to the induction field around the line (because it serves as a waveguide), there are effects due to the finite length of the transmission line. 14

In Canada, concern that fields from power line carrier may interfere with the signal from aeronautical nondirectional navigation beacons-with potentially disastrous consequences-has prompted attention to power line carrier emissions from transmission lines. Canadian power line carrier systems that operate in the ranges 30-200 kHz and 425-490 kHz have not required licenses since 1983, and can operate in any band on a non-interference basis. The radiation

characteristics of power lines with power line carrier are largely unknown, because so little work has been done, and there are no standards except those that certain electric utilities have developed for their own use.

The Canadian Electrical Association therefore initiated a project to quantify power line carrier emissions.15 Among the findings from this project were that the parallel field was stronger than the perpendicular field out to 200 meters from the line. As one moves laterally away from the transmission line, the fields drop off sharply, though in somewhat different ways. Distribution lines can cause up to 20 dB signal variation up to 200 meters away. Ambient noise levels tend to mask power line carrier emissions past 1500 to 2000 meters from the transmission line.

Line noise has been an impediment to the use of power line carrier on distribution circuits, especially with respect to the building automation market. 16 The 5-1 00 kHz band of frequencies is used for carrier communications on distribution lines in the USA. The term "distribution line carrier" refers to a system in which data transmitters and receivers are located at many points within the power distribution network, often within the buildings served. 1 7 Effort has been devoted to characterizing the properties of these circuits, to facilitate design of distribution line carrier systems.' 8

Power line carrier has historically been little affected by regulatory issues. This makes distribution line carrier attractive to the building automation industry, now that the problem of line noise has been overcome by the application of spread-spectrum technology to power line carrier, and the Consumer Electronic Bus standard has been widely accepted. 16

There do exist some sources of interference on building wiring, though. These include power supplies for some halogen lights (which produce strong harmonics above a 25-kHz fundamental frequency), switching power supplies (which produce harmonics above 50 kHz) and in homes, certain brands of baby monitors (I 00-300 kHz). 16 Television receivers may also contribute.

In summary, in the USA power line carrier was used mostly on transmission lines and only sporadically on distribution lines throughout the middle part of the twentieth century. However, in the 1980s, applications for the use of carrier current on distribution systems and on building wiring began to multiply rapidly, so that it is now used on many distribution systems and by some devices that are used in homes or offices.

Power line carrier has been declared to be safe by the electric power industry, but this claim does not appear to be based on the results of any scientific or experimental studies. So far as I have been able to discover, the RF fields present around electric wires as a result of the presence of power line carrier on those wires have never been evaluated with respect to their healthfulness. (Perhaps they have *been presumed* to be safe, based on existing RF health protection standards, which I have criticized as being inadequate to protect health; see Part 1, Section 3 of this document.)

Two Italian scientists published a scientific paper 19 nearly four years ago calling attention to the presence on high- and medium-voltage electric power lines of radio-frequency currents, pointing out that these currents imply the presence of an RF field in the vicinity of the lines, and there is a need to evaluate the health hazard implications of the presence of these RF fields. They further commented that, although the intensity of these RF fields is low, "the intensity of currents induced in the human body by exposure to magnetic fields increases with frequency."

What they meant by this statement is that the strength of an induced current depends directly on the frequency of the inducing field. Thus, if two fields are equally strong, but one has a higher frequency than the other, the field of higher frequency will induce a stronger current than the other field, and ratio of the strengths of the induced currents will be the same as the ratio of the frequencies of the inducing fields.

The actual hazard to health from the non-ionizing portion of the electromagnetic spectrum seems to arise from a flow of current through living tissues. Such current may flow either by direct contact with electrodes having a difference of potential, or-if one is exposed to a field in air---by induction. Vignati and Giuliani were simply making the point that, because of their high frequency, exposure to RF fields in air can be hazardous, even when the strength of the field is extremely small, because of the multiplier effect of the field frequency on the magnitude of the current it can induce.

More recently, a retired professor of electrical engineering has also urged 6 that attention be paid to the presence of radio frequencies on electric power systems, and their implications for human health.

Section 3. WhatconstitutesavalidEMFexposuremetricforhealthprotectionpurposes?

The strength of the electric or magnetic field is the customary exposure metric used in studies of the health effects of exposure to such fields.

Being suspicious that existing exposure metrics were inadequate, I undertook a theoretical investigation of the question: What constitutes a valid metric for exposure to a non-ionizing electromagnetic field, or to non-ionizing electromagnetic radiation, for health protection purposes? My findings to date have been published in a symposium volume 20 on cellular telephone health hazards. Because the equations I derived are valid at all frequencies, they are applicable to the fields around electric power lines.

What I learned is that there are *two* exposure metrics---not one---for the non-ionizing electromagnetic field. One metric is appropriate for protecting against thermal hazards to health; the other is appropriate for protecting against nonthermal hazards to health. The choice of which exposure metric to use in any given situation depends on what kind of health effect is of interest.

Cancer is a *nonthermal* health effect. 2 1 Therefore, when carrying out epidemiological studies on cancer and electromagnetic fields, one ideally ought to be employing an appropriate *nonthermal* exposure metric. This is not possible at the present time for fields that do not have the configuration of a plane wave, because a practical exposure metric for the type of field that exists around an electric power line has not yet been determined (as is discussed below). Other parameters can be employed, though, such as the distance from the current-carrying wire (also discussed below). Indeed, the wire code of Wertheimer and Leeper is a fairly good "nonthermal exposure index", as the findings of the Savitz study5 demonstrated, when it was discovered that the disease cases correlated better with the wire code category than with any other environmental parameter.

I also learned that the existing standards for protection against exposure to RF radiation, which employ the radiation intensity as an exposure metric, and impose an upper limit on this value for health protection, use an exposure metric that is theoretically valid only for *a plane electromagnetic wave,* and impose an upper limit that protects only against a *thermal* hazard.

I have derived and published 20 perfectly general point-function equations for thermal and nonthermal exposure metrics, but these are not useful, practical exposure metrics. I derive the usual exposure metric that is in common use today by integrating the point-function metric over time and a finite volume of space in a Cartesian coordinate system, appropriate for a plane electromagnetic wave, thereby proving that the radiation intensity under plane-wave conditions can be employed as a valid metric for thermal exposure.

It is much more difficult to carry out a derivation of a practical exposure metric for nonthermal health hazards. However, since publishing my cited work, I have satisfied myself that, when the exposure is to a plane electromagnetic wave, the radiation intensity also is a valid metric for nonthermal exposure, although the "safe limit" values will be different.

Radiation intensity can be obtained as a product of the strength of electric and magnetic fields. This is the reason why it has been assumed that the strength of such a field is an appropriate exposure metric. However, as I point out here, there are stringent limitations on the validity of this assumption. The fields around electric power lines do *not* take the form of plane waves, so the use of field strength as an exposure metric is *not* valid for either the electric or the magnetic field around power lines.

When exposure is to a field that does *not* have the form of a plane electromagnetic wave, then the exposure metric is a complicated function of field variables, and the field strength alone does *not* constitute an adequate exposure metric for health protection or biological study purposes. This is the situation that exists in the vicinity of electric power lines, at all frequencies.

I have learned one more important thing, documented in my published paper: 20 while the "safe limit" for protection against a thermal health hazard (in the case of a plane wave) consists of an upper limit, the "safe limit" for protection against a nonthermal health hazard (in the case of a plane wave) very likely consists of both upper and lower limits. The reason is that nonthermal health hazards seem to appear within a "window" of frequency and intensity values; the purpose of "safe limits" is then to ensure that one is *outside* this "window of hazard". One must either be below a low "upper limit" value, or above a high "lower limit" value.

If this sounds confusing, realize that the purpose of the "safe limits" is to keep people "out of the window", so that they are *not* between the upper and the lower "safe limit" values. For safety, they must be either above the high value (which is then serving as a lower limit of safety) or below the low value (which is then serving as an upper limit of safety).

While studies have been carried out that might permit an evaluation of these "safe limit" values,
I have not reviewed the scientific data in sufficient detail to do so.

The concept of a "window of hazard" very likely applies to *all* fields, not just to plane waves. So I would expect that, in general, the concept of two "safe limit" values will apply whenever health effects of a nonthermal character are of concern.

Having said that, I should point out that for *complete* protection, one wants to be safe from *both* thermal and nonthermal health hazards. This will be most easily and conveniently accomplished by complying with whichever upper limit is *lower.*

For a plane electromagnetic wave, it is clear that the upper limit for protection against a nonthermal health hazard will be considerably lower than the already-established upper limit for protection against a thermal health hazard. What this means, unfortunately, is that compliance with existing radio-frequency health protection standards provides no guarantee of safety.

I have studied, theoretically, the characteristics of radio-frequency fields in the space around a long straight wire; in other words, the characteristics of RF fields around electric power lines. Their intensity falls off *very* strongly as the perpendicular distance from the wire increases; this means that it is possible to get far enough away from the current-carrying wire to be able to disregard the RF field that surrounds it. In other words, there *is* such a thing as a "safe distance" from a power line carrying RF current; the RF health hazard will depend strongly on the distance from the power line.

I think this explains the "wire code paradox": why the wire codes of Wertheimer and Leeper correlated so much better with disease cases than did measured 60-Hz magnetic field strength. To begin with, the device that Ed Leeper made for Nancy Wertheimer, which she used in lieu of a gaussmeter to "probe" the fields around the distribution **wires,** would have responded much more strongly to RF fields than to 60-Hz fields (because the response depends on

frequency, and RF frequencies due to power line carrier, for example, are likely to be more than one hundred times higher than the fundamental frequency). Based on the description given to Paul Brodeur4 by Nancy Wertheimer, I believe her detector was responding to an RF field around the distribution wires, but she and Ed Leeper believed it was responding to a 60-Hz field, and wrote their paper accordingly.

I have tried to confirm the presence of RF currents on Denver distribution lines, without success to date. I nevertheless believe that the wire codes developed by Wertheimer and Leeper were based on their unknowing detection of RF fields around electric distribution lines, and that the explanation of the "wire code paradox" is that these wire codes served as an excellent surrogate for the RF fields around electric power lines that have never been measured, but which in my professional judgment are the cause of the human disease and illness associated with electric power lines in the Risk Evaluation Report of the California EMF Project (and in published scientific paper and reports).

More research needs to be done to develop a scientifically sound exposure metric for nonthermal health effects. However, the simple "magnetic induction current detector" that Nancy Wertheimer used, which responds to dB/dt, is a practical device that, if calibrated, could be used to quantitate hazard around electric power lines. The results from it ought to correlate quite well with health risk, probably even better than wire code categories do, because it directly measures the hazardous effect of alternating current magnetic fields: their ability to induce a flow of current in a material medium. (However, because the toxicity to living tissue of electric current flow depends on the frequency of the current, an instrument that can respond selectively to different frequency bands would be most useful for scientific investigation at this time.)

At present, we know the exposure metric only for a plane electromagnetic wave: the radiation intensity. Scientific formulas can be written to express the exposure metric in mathematically correct form; but practical, useful exposure metrics for non-plane electromagnetic fields and for low-frequency, separable electric and magnetic fields (and instruments for making measurements of them) do not yet exist. Further research is needed.

Section 4. Some historic facts* about *electric power distribution systems in the USA

There are two basic types of distribution system design: the delta (symbolized by **A)** and the wye (symbolized by Y). There are three phases of electricity that come from a generator, designated A, B and C. The current and voltage of any one phase is out of phase with the other two by one third of a cycle: 120 degrees. Each phase requires a separate wire, so the minimum number of wires required for transmission and distribution systems is three.

The delta-configured distribution system employs three wires. Each wire carries current for one of the three phases, and return current in the opposite direction for a different phase. All wires are maintained at an electric potential elevated above the potential of the earth, so none of the system wires is electrically grounded.

The wye-configured distribution system employs four wires. Three of the wires are maintained at an electric potential elevated above the potential of the earth, and carry current for one of the three phases. These wires are called "phase conductors". The fourth wire, called the "neutral wire", carries return current for all three phases and is maintained at ground potential, which is accomplished by the presence of one (or more) electrical connections with the earth.

A wye-configured distribution system may be either single-grounded or multi-grounded. If the system is single-grounded, the neutral wire is connected to the earth at only one point along the distribution line, usually at the substation. If the system is multi-grounded, there will be ground connections from the neutral wire at multiple points along its length, at certain of the poles that support the wires.

In the USA in the early 1920s, all distribution systems employed the delta configuration. This means that there were no electrical grounding connections from these wires to the earth. The probable reason why the delta configuration was preferred was that power companies wanted to keep costs low, and the delta system required only three wires, while the Wye configuration required four wires. Therefore the delta configuration was the one with the lowest capital cost. (Electric companies were stringing a lot of new lines at that time, to serve new customers, so keeping capital costs low was a major concern.)

Today, virtually every distribution system in the USA is of the multi-grounded wye type. (There are a few of the old delta-configured distribution systems still in existence.) A massive changeover took place in electric companies across the USA, beginning in the late 1920s or early 1930s. By the 1950s, this was probably pretty nearly completed all over the USA.

Why did this change-over occur? To save the power companies money. They discovered, in the late 1920s or early 1930s, that using a multi-grounded wye distribution system would allow electric current that properly should flow on the neutral wire to flow on the earth. A single-grounded wye system does not offer this "benefit" because it does not connect the earth in parallel with the neutral wire, but a multi-grounded wye system *does* connect the earth in parallel with the neutral wire, so the current that would otherwise flow on the neutral wire will divide and flow in part on the neutral wire, and in part on the earth.

We know that the motive was to save money because a paper 22 published in 1956 says so. It may be difficult for the reader to understand how a power company could save money by replacing a three-wire system with a four-wire system. I have found no explicit explanation, but I may have been able to figure it out.

The wye-configured distribution system employs a single wire-the neutral wire-to carry the return current for *all three* phase conductors. The current on the phase conductors is displaced by one-third of a cycle from one phase to the next. (There is no standard order for the direction of this displacement, in terms of the notation A, B and C.) On the neutral wire, the current from all three phases is present in combination. If this is pure 60-Hz current, and if the current is balanced between the three phases, so that the same current is flowing in all three phases, then the sum of the current from all three phases is zero.

In a real system, of course, the balance won't be exact, so the total current flowing on the neutral wire won't be *exactly* zero. But there will be enough cancellation so that the total current will be *nearly* zero. This means that the neutral wire will carry much less current than any one of the phase conductors, so a smaller-diameter wire can be employed for the neutral wire, compared to the wires needed for the phase conductors.

This is the way the earliest wye-configured distribution systems were set up. But the foregoing analysis is valid only for electric power of very good quality, where virtually I 00% of the current in the distribution system is at the fundamental frequency.

Now we need to look at what happens when the quality of electric power deteriorates somewhat, because of the presence of low harmonics on the distribution system. The third harmonic is particularly important, because it completes a full cycle in the time that the fundamental completes only one-third of a cycle. The same phase difference between any two of A, B and C that is one third of a cycle for the fundamental frequency is a full *cycle* for the third harmonic! The result is that on the neutral wire, where current from all three phases is combined, any third harmonic current that may be present is the *sum* of what is present on the separate phase conductors! Assuming a balanced system, there will be *three times* as much third harmonic current on the neutral wire as on any single one of the phase conductors! The same is true for all odd multiples of the third harmonic.

Clearly, if the distribution system current has a good deal of harmonic content, the neutral wire is going to be carrying *more* current than the phase conductors, at some of the harmonic frequencies. This means that the neutral wire may need to be *larger* in diameter than the wire for the phase conductors!

Notice how the needed size of the neutral wire changes, depending on the quality of the power in the distribution system. When the power quality is very good, a small neutral wire is adequate; but if the power quality deteriorates because of an increase in the presence of harmonics, the size of the neutral wire needed can climb dramatically.

Large-diameter wires are not only more expensive than small-diameter wires (the mass increases as the square of the diameter), but they are heavier; they may require stronger supporting structures; and they may require a different degree of "sag". So there are some sound reasons why a power company that had installed the wires for a wye-configured system when power quality was good, might want to avoid replacing the small neutral wire with a larger one after it noticed that the quality of electric power on its distribution system had declined, and much more current was travelling on the neutral wire than the system designers had foreseen.

A very inexpensive way to deal with such a situation would be to let some of the cur-rent from a now-undersize neutral wire flow on the earth by grounding the neutral wire at a number of points along its length, so that the earth would be electrically connected in parallel with the system's neutral wire. Installing some grounding connections would be very cheap, quick, and easy to do, compared to replacing the neutral wire with one of larger diameter.

Of course, converting delta-configured systems to multi-grounded wye systems would involve some expense. But if power quality had deteriorated, the existing delta-configured systems were probably carrying more current than the designers had anticipated, and were going to require upgrading much sooner than had been anticipated. So it is not surprising that an evaluation of the economics of the situation led virtually all electric power companies to commit to making the change from delta-configured to multi-grounded wye distribution systems in the 1930s.

The only thing missing from this scenario is evidence that there actually *was* a deterioration in the quality of electric power across the country between the early 1920s and the 1930s. At this time I have no proof, but I do know that the first license for commercial radio broadcasting was issued in November, 1920, to KDKA in Pittsburgh, PA, and this set off an explosion of commercial radio broadcasting across the entire USA. By the end of November, 1922-just two years later-there were 536 commercial stations licensed and broadcasting radio programs !23

In order to listen to these broadcasts, people had to have radio **receivers.** There was an explosion in the manufacture and sale of radio receivers for home use, all across the USA. These radio receivers had to be powered, and while some operated from batteries, many used electricity from the convenient electrical outlet in the wall at home.

These early radios used vacuum tubes, which drew quite a bit of current, by today's standards, and which would have constituted the first electronic, nonlinear electric load in mass use in the home by ordinary citizens-in contrast to industrial loads-in the USA. The flood of these radio receivers into the homes of electric customers all across the USA represented the first mass influx of a decidedly nonlinear electric load on electric power circuits in the history of the electric power industry in the USA. And it happened very rapidly---in less than a decade---between the beginning of 1921 and, probably, the beginning of 1926.

It seems likely that the mass influx of radio receivers as a new type of load on electric power systems across the USA during the 1920s-a load that I hypothesize introduced a substantial harmonic content into household electric wiring and also onto the distribution wiring, producing a decided deterioration in the quality of electric power-caused the electric power companies to abandon a distribution system configuration that had been perfectly satisfactory in 1920-the ungrounded delta-configured system-and by the late 1920s, to decide to replace it with a multi-grounded wye

distribution system that permitted some electric current from the distribution system's neutral wire to flow over the earth.

There does not seem to have been any harm caused by this flow of electric current over the earth at the time it first occurred. But it set the stage for a problem that began to appear in the 1980s, after there had been a long period of further deterioration in the quality of electric power. This problem first appeared on fan-ns, where it is now widely known as "stray voltage". [The term “stray voltage" has a legal definition in most states, relating to its perception as a simple problem of 60-Hz electrical grounding. But there is reason to believe it is actually caused by RF current on the earth, present because of long deterioration in the quality of electric power on power systems, which has now reached a point where it is actually causing harm, because the frequencies and RF current strengths have gotten so high.]

Section 5. A comment about waveguides

Our electric power system uses wires to transmit electrical energy from one place to another. One view is that the electric current flowing in the wires transmits this energy, but a view more consistent with electromagnetic field theory is that the energy is transmitted in the dielectric medium surrounding the wires-the air-and the wires simply serve to *guide* the waves that travel through the air (much as railroad tracks determine where a train goes).

The electric wires of a transmission or distribution system are known as an "open waveguide system" because the EMF surrounds the wires. It is also possible to have a "closed waveguide system" where the EMF is confined inside a hollow electric conductor. Closed waveguide systems are used for the transmission of microwaves, for example.

In an open waveguide system, people can be easily exposed to the EMF around the current conductor. In a closed waveguide system, this is impossible (unless a leak-an opening in the waveguide---develops). So closed waveguides are safer than open waveguides.

If electric current were confined to the wires of the electrical system, then these current-carrying wires would be the only waveguides in the electric power system. It would be possible to keep one's distance from most of them.

When electric current is allowed to travel over the earth, then those portions of the earth carrying electric current from the power system become part of the open waveguide system used to transmit electrical energy from one place to another. How can one keep away from the earth, when the force of gravity keeps us in continual contact with the earth's surface? Birds can **fly** away, but most terrestrial animals can only try to relocate to some other patch of land that hasn't become part of some electric power company's open waveguide system.

From the 1930s to the present, current from electric distribution systems has been flowing over the earth. There is no clear evidence that it adversely affected human health during the decades from 1930 to perhaps the 1970s. It would seem that, so long as the frequencies and the current strengths were sufficiently low, animals that live on the surface of the earth were not harmed by the use of the earth as part of the open waveguide system used to transmit electrical power.

But the frequencies didn't stay low, because the quality of electric power has been steadily deteriorating over time, due to our increasing use in homes, offices and businesses of electrical equipment that constitutes a non-linear load.

As the quality of electric power has deteriorated, and the frequencies present on electrical wiring and electrical distribution systems have climbed, the portion of the earth's surface carrying electric current from distribution systems has slowly and gradually become part of an open RF waveguide. This means that, as time passed, people and animals in locations where there is power system current on the earth have found themselves living on the surface of an open RF waveguide.

From laboratory studies of the health of small mammals inside closed RF waveguides, it is very well known that the active interior surface of an RF waveguide is hazardous to health. It can be expected that living on the active exterior surface of an open RF waveguide will be hazardous to mammalian health, also.

Section 6. Contact current. A health hazard from electric power system current on the earth

When an animal is in contact with a surface over which current is flowing, a current can flow through a portion of the animal's body, if there is a potential difference between any pair of contact points. Consider a person standing on a surface over which a small current is flowing, with his feet approximately one foot apart. If there is a non-zero difference of potential between his two feet, then current will flow through his body between his two feet. Very roughly, the path of current flow will be through each foot and leg, and through the lower portion of his trunk, where it might affect some organs in his lower abdomen.

If this person performed a hand-stand, then the current would flow through his two hands and arms, and through the upper portion of his trunk, where it might affect his heart.

Because of the "window of hazard" concept already discussed in Section 3 in conjunction with nonthermal health effects, there is reason to expect that there is a certain range of tissue current density that is harmful. Tissues experiencing a current density within this "window" could be injured, while tissues experiencing a very different value of current density-either higher or lower-would escape injury.

Consider, now, a four-legged creature, such as a cow. There are six different possible contact current paths through the body of a cow, if its four legs are considered in pairs: (1) between the two front legs; (2) between the two rear legs; (3) between the legs on the left side; (4) between the legs on the right side; (5) between one diagonally opposed pair of legs; and (6) between the other diagonally opposed pair of legs. Because current can flow simultaneously through all these paths, there will be *more* current passing through the animal's trunk than consideration of any one cur-rent path would indicate.

If we compare a four-legged cow with a two-legged human being, and assume that the tissue current density that is harmful to mammalian health has the same values in both species, then it can be seen that a smaller flow of current through a single pair of legs is likely to be harmful to the internal organs of the cow, than to the internal organs of a human being, because the cow has *six* paths through which current can flow simultaneously through the trunk of its body, while the human being has only *one* such path.

Therefore, if a harmful current is flowing over the earth and is slowing getting stronger as time passes, it would be expected that a cow would be affected sooner-at lower currents-than a human being would.

This is exactly what has been observed on dairy farms that have a "stray voltage" problem.

States perceive "stray voltage" to be a problem of poor electrical grounding, and have defined it in terms of a voltage difference at 60 Hz. Where cows are affected on a dairy farm, the voltage difference is measured between "cow contact" points: a pair of locations that a cow might touch at the same time, allowing a current to flow through its body. The state's definition presumes that the current flow giving rise to the problem is a 60-Hz current.

An early effect on dairy cows was a reduction in their milk production. This damages the farmer economically, so it was taken seriously by state officials everywhere.

It is known that milk production is a function of the amount of water that the cow drinks.24 So any phenomenon that might motivate a cow to drink less water could be expected to lower its milk production.

Water tanks from which cows drink are often made of metal. If there were a difference of potential between the earth where the cow is standing and the metal of the tank, where the cow's head or neck will touch it when it attempts to d@, then there is the possibility that the cow could get a mild electric shock from 60-Hz current which might be unpleasant enough to cause an aversion on the part of the cow to drinking water, unless it was very thirsty.

This seems to have been the original basis for associating "stray voltage" on a dairy farm with a 60-Hz current flow between two cow contact points where there is a difference of potential at 60 Hz. However, the health problems on farms with a "stray voltage" problem are not always eliminated by reducing 60-Hz voltage differences, as the states now require.

Here are a list of health problems or biological phenomena that have been reported on farms with a "stray voltage" problem. Beside each is a notation regarding the scientific evidence that this phenomenon or health problem is associated with (1) 60-Hz current in the body, and (2) RF current in the body (including exposure to RF fields, where it is presumed that an induced current will flow at the field frequency). It is assumed that the biological effects will be similar for all mammals, so that evidence across different mammalian species-specifically, mice, cows and human beings-can be combined.

Cow Health Effect or Biological Phenomenon	60 Hz	Radio Frequencies
Difficulty conceiving when bred	none	3 epidemiological studies of reproductive outcome in female physiotherapists 25-29
Reduced milk production	see foregoing paragraph beginning: "Water tanks	phenomenon of electrodessication 30
Necrotic lesions on body or legs	none	necrosis observed in mice in a laboratory experiment where they were exposed to a VHF RF field in air"

A careful review of the scientific evidence concerning the health effects of exposure to 60-Hz fields has been conducted in both the USA' and Japan 2 but no evidence of interference with human reproduction has been found. But three epidemiological studieS25-27 of female physical therapists who are occupationally exposed to radio-frequency radiation have been carried out, and there is clear evidence in two of them of reproductive problems: in one, miscarriage; in the other, fetal abnormality. (One of them prompted an exchange of letters 28-29 in which the point was made that theory ought to explain experimental results, instead of experimental results being adjusted to "fit" a particular theory.)

No studies of milk production have been carried out in any mammalian species other than dairy animals. All such studies that investigate the effects of electricity have addressed 60-Hz current. Many of these studies show little or no adverse effect of the 60-Hz current on animal health or on milk production.

There is a phenomenon known as *electrodessication,* which is defined as "the drying up of tissue by use of a high-frequency electric current applied with a needle electrode". 30 If the mode of application of the current to the tissue is irrelevant, as seems likely, then electrodessication would be expected to occur as a consequence of passage of an RF current through living tissue. It is already known that milk production requires adequate water in the body,4 so it

is reasonable to expect that a lactating mammal experiencing an electrodessication effect from RF current passing though its body would display a reduced output of milk.

The phenomenon of electrodessication in laboratory mice subjected to a high-frequency RF filed in air has been reported.31

On farms with a severe "stray voltage" problem, cows may develop necrotic lesions on their legs or bodies. There is no known association of such lesions with exposure to 60-Hz current, but necrosis of the ears of laboratory mice exposed to a high-frequency RF field in air has been reported.31

In summary, for each of the three biological phenomena listed above, exposure to RF current is much better supported by the available evidence as a possible cause of the biological effect than is exposure to 60-Hz current. When a common cause is sought for *all three effects,* it becomes obvious that RF current passing through the body of a cow is a *much* more probable cause than is 60-Hz current, because there is far more scientific evidence to support it!

Therefore the cow health problem on farms called "stray voltage" almost certainly results from exposure of the tissues of the cow's body to RF current on the earth, which comes from electric power systems. (It is not known exactly which RF frequencies are harmful, but the manner in which the problems of "stray voltage" have made their appearance over time is consistent with an increase in the RF content of electric power occurring because of progressive deterioration in the quality of electric power over a period of decades. Therefore, it would appear that the problem can be corrected by improving the quality of electric power by removing RF from the electric power system.)

Section 7. Environmental hazards

The flow of current from the electric power system over the earth doesn't flow evenly, because the electrical resistivity of the soil differs from place to place. (This is why not all the farms in a given area are equally affected with "stray voltage" problems.) Moist soil is more electrically conductive than dry soil, so the electric current on the earth tends to concentrate in certain locations, including in creeks and other streams of water.

Fish and amphibians, such as frogs, live in such streams and creeks. When there is electric current in the water from the electric power system, it can be expected to kill them, or possibly to produce developmental abnormalities.

No studies have been done, yet, to relate the health of aquatic animals to the amount and the frequency of electric current in their aquatic environment. But there has been a sufficiently serious effect on terrestrial mammals to anticipate that such studies, if performed, would show that the very small RF current from electric power lines exerts either a lethal effect, or gives rise to developmental abnormalities.

Another fact worthy of note is that the RF current on the surface of the earth from power lines is not confined to soil and water; it spreads over everything that is in contact with the earth, including the floors and other surfaces of buildings, the surfaces of furniture inside buildings, and trees and vegetation that is growing on the earth. In an affected region, birds will experience it while roosting in trees. People will experience it while asleep in their beds.

The easiest way to escape this current is to change one's geographic location. Property owners often find it uneconomic to do this.

It is not impossible to escape its biological effects while remaining on a property where it is present, but to do so is usually prohibitively expensive.

Section 8. A comment about the U.S. Congress and the federal government

The U.S. Congress has contributed substantially to the increasingly rapid deterioration of power quality on building wiring, and probably also on distribution system wires, over the past quarter century by the legislation it passed in the 1970s mandating the use in the USA of energy-efficient electrical appliances. This legislation made it illegal to manufacture, import or sell certain types of energy-inefficient appliances that had been in common use up to that time.

Unfortunately, the way energy efficiency is usually achieved in the design of an electrical appliance is to draw current in a large number of short pulses, rather than steadily and uninterruptedly, as energy-inefficient equipment did. Drawing current in brief pulses generates high frequencies (as was mentioned in Reference 1, page 24). So replacing an old, energy-inefficient electrical appliance with a new, energy-efficient one replaces a linear load with a nonlinear load! This will cause a substantial deterioration in the quality of the electric power on the building wiring, and because these high frequencies are likely to escape out onto the distribution lines that deliver electric power to the building, they will deteriorate the quality of electric power on the distribution lines.

While Congress made it illegal to sell energy-inefficient electric appliances, there were a great many already in use. Fluorescent lamps, in particular, have a long life-nearly a decade-and are normally replaced one at a time, as they bum out. It would have taken a decade to replace all the fluorescent lamps in a building, at normal replacement rates.

To speed up their replacement with the new, energy-efficient designs, the U.S. Environmental Protection Agency (EPA) conceived of a program to speed up replacement of fluorescent lamps in schools: for those schools that agreed to participate, the EPA would pay the cost of purchasing new fluorescent lamps so that an immediate replacement of all fluorescent lamps in the building could be carried out. A number of schools have accepted this offer, because the new design of fluorescent lamp uses less electricity, and accelerating the replacement would lower the school's electricity bill.

Unfortunately, the most energy-efficient design of fluorescent lamp not only puts RF on the wiring of the building, as described above, but it also emits RF radiation into the space around it! Thanks to the U.S. EPA, many schools in this country have now been turned into RF radiation chambers, where there are hazardous RF fields not only around the building wiring, but also beneath the overhead lights in the classrooms!

Electric power companies have also joined the energy-efficiency crusade to rush replacement of fluorescent lamps by offering reduced electric rates to their large, institutional customers who commit to prompt, wholesale replacement of the old, energy-inefficient designs with new, more energy-efficient designs.

These programs have greatly accelerated the deterioration of power quality in buildings, and also probably on distribution systems. They probably have done more than anything else to increase the amount of RF on building wiring and electric distribution lines to a level that is hazardous to health and that is currently causing ill health, and sometimes frank disease, in human beings today.

Without realizing or intending it, the U.S. Congress mandated that U.S. citizens must abandon a lifestyle that was healthful, and replace it with one that is unhealthful, when it passed legislation mandating energy efficiency a quarter-century ago.

Section 9. Summary comment on the Risk Evaluation Report

The Risk Evaluation Report identifies real health risks that most certainly need to be taken seriously and addressed effectively, but is mistaken in attributing the cause of these risks to 60-Hz fields.

The totality of the available evidence allows only one conclusion: the true cause of these health risks is the presence of radio-frequency currents on electric power systems, and the RF fields they produce in the space around current-carrying electric wires. In some cases the RF currents have been deliberately introduced, but more often they are unintentionally present as the result of the widespread use by electric power system customers of nonlinear loads, which has contributed to a deterioration in the quality of electric power.

Part II. Comment on Policy Implications

Because exposure to 60-Hz fields is *not* the cause of the health problems associated with electric power lines and electrical wiring, there is no health benefit to be gained by reducing the strength of any 60-Hz fields. This means that putting 3-phase power lines underground will not be beneficial to health, for example. On the contrary, if there is RF on buried wires, the hazard to human health will be *increased* by burial of the lines, because then people can get closer to the current carrying wires, where the RF field is likely to be most hazardous.

A strategy of improving power quality by (1) removing RF from electrical wiring, and (2) preventing RF from getting on electrical wiring will be effective. An effort in the state of Wisconsin to accomplish both is under way at this time.

Capacitors can be connected to building wiring, between the "hot" and neutral wires, at the point where electricity enters the building, as a way of preventing RF on building wiring generated by appliances from getting out onto the distribution lines. If every electric company customer had such an RF filter at the point of electricity delivery, the harmful RF pollution of distribution lines by customers would be largely prevented, and the quality of electric power on the distribution system would be much improved. (Of course, the power company would need to address its own contribution to poor power quality, such as are caused by the switching of substation capacitor banks. And an RF "trap" may be needed at the interface between transmission line and distribution system, to prevent one from "polluting" the other.)

The use of capacitors to control RF on power lines is well-known. They may be passive (as in the suggestion for application to building wiring in the foregoing paragraph) or active.32

Legislation will be required to change the design of electrical equipment, so that it doesn't put high-frequency current on building wiring. Such legislation is being developed in Wisconsin at this time. A draft version is expected to be ready for review in October, 2001.

I recommend visiting the Web site <www.toxicelectricity.com> to monitor news from Wisconsin on this topic. The site is updated weekly.

The installation of capacitors on household wiring has been anecdotally reported to reduce the symptoms of people suffering from chronic fatigue syndrome. A copy of a story in a Wisconsin weekly newspaper telling of this is included as Appendix C.

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5. D. A. Savitz, H. Wachtel, F. A. Bames, E. M. John & J. G. Tvridk. Case-control study of childhood cancer and exposure to 60-Hz magnetic fields. **American Journal of Epidemiology 128** (No. 1; July 1988) 21-38.
6. Martin Graham. A Ubiquitous Pollutant. University of Califomia-Berkeley Electronics Research Laboratory Technical Memorandum 55, October 2000.

E-MAIL 1

Jack, this message is to identify what I believe to be a spelling error in the draft document "An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances".

First, I have to identify where the misspelled word is: in Appendix Two (titled "Electric and Magnetic Fields Risk Evaluation Guidelines").

This Appendix Two is a full paper in its own right, with its own appendices. The misspelled word is in the title to this second Appendix Two (which is titled: "How to Express Quantitively ...").

The word I think is misspelled is "Quantitively"; I think the intended word was "Quantitatively". -- Marjorie Lundquist

E-MAIL 2

Jack, in my e-mail message earlier today about the misspelled word in the draft document, I forgot to provide a page number.

The misspelled word is on page 49 of Appendix Two (titled "Electric and Magnetic Fields Risk Evaluation Guidelines"). -- Marjorie Lundquist

E-MAIL 3

Jack, this is a preliminary comment on the draft document titled "An Evaluation of the Possible Risks From ELectric and Magnetic Fields ..." being made by e-mail for speedy transmission. (I am preparing a written response which will be mailed.)

I have been working to found a new discipline called "bioelectromagnetic hygiene" which is relevant to the question of the healthfulness of the fields around electric power lines, and also the fields around cellular phones and their base transmitters. The first publication relating to bioelectromagnetic hygiene came out in October, 2000. It is Chapter 4 in the symposium volume titled "Wireless Phones and Health II: State of the Science" (George Carlo, ed.; published by Kluwer Academic Publishers).

Chapter 4 addresses the question of the exposure metric appropriate for health hazard evaluations, providing a theoretical derivation from the fundamental theory (Maxwell's equations of electromagnetism). Although this book addresses the microwave fields of wireless radio-telephone technology, the fundamental physics is independent of frequency, and the information about exposure metrics is therefore generally applicable to all electromagnetic fields, including those around electric power lines.

In the written commentary I am preparing for mailing, I am discussing the contents of Chapter 4, since this is a published work.

I am sending you this "advance notice" by e-mail in order to enable the California Health Department to move promptly to purchase this book (if it has

not already done so in relation to its efforts to address the health hazards of cellular phones and radio-telephone technology generally).

Protecting the health of people from the harmful effects of exposure to electromagnetic fields is a bioelectromagnetic hygiene task. For this reason, published literature on bioelectromagnetic hygiene is pertinent to the mission of the California Health Department, and is especially pertinent to the task addressed by the draft document upon which comment from the public has been invited.

When the book cited here has been acquired, and when my written comments are received and reviewed, I think the California Health Department will want to include Chapter 4 of this book among the documents referenced in the bibliography of this report. -- Marjorie Lundquist

To: Jack Collins, Calif.EMF Program
From: Wayne Lusvardi
Re: EMF Review Study

I have reviewed the California EMF Study and find it duplicative of other studies, unbalanced, and lacking in common sense. The report fails to tell the public that exposure to radiation from sun at the beach is an exponentially larger danger than EMF's from man-made electrical transmission lines, wiring in buildings, or appliances. The study fails to tell how the life span of the average person may be shortened without the conveniences of electrical systems. The statement in the "Conclusions" section of the draft report that "...there is a chance that EMF's have no effect at all" is a gross understatement. There is OVERWHELMING evidence that EMF's have no effect at all. How much money was spent on this slanted and redundant study? What is the real political agenda of the study? More frivolous lawsuits for trial lawyers? I advocate for de-certification of the study as bogus.

Wayne Lusvardi
330 Pleasant Street
Pasadena, CA 91101

Nonlinear Technologies, Inc.
PO Box 7284, Long Beach, CA 90807 * 562-426-1639

July 28, 2001

Jack Collins
Califomia EMF Program
1515 Clay St., Suite 1700
Oakland, CA 94612

Dear Mr. Collins:

As a citizen of California, I am dismayed that the state is still spending money on EMF "research." As a scientist, I am appalled by the report that you are considering inflicting on the public. This kind of thing might have been marginally justified ten years ago, but today it is complete hogwash. It never should have been produced.

I can't begin to enumerate the things that are wrong with it. It treats ancient, discredited research as valid. It uses lofty-sounding but nonsensical technobabble such as "resonance between 60 Hz magnetic fields and the earth's magnetic field" and "rephasing" of power lines. And so on. More than I can enumerate in one letter.

It astonishes me that you would consider influencing public policy with a report based on the opinions of only three researchers, while completely ignoring the conclusions of the National Academy of Sciences and the National Cancer Institute. These massive studies, by hundreds of the best scientists in the country-and of which your pitiful three reviewers seem totally ignorant-concluded that there is no link between any form of cancer and power line magnetic fields. One would think that such a definitive conclusion would put the matter to rest. The great State of Califomia, however, seems to want to side with the fearmongers.

Sincerely,

Stephen Maas, Ph. D., P. E.
Former Professor of Electrical Engineering, UCLA President, Nonlinear Technologies, Inc.

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Dear Raymond, please find enclosed my comments to the document By the California EMF Program. I focused so far on the general statements and on the leukemia and brain tumour sections. The leukemia is in my opinion the most important since the most important evidence of association regards this disease. The work done is really enormous: how long did it take? I will continue to review the document and I will send my new comments in ten days.
Best regards, Corrado Magnani

General comment
The document presents in great detail the process adopted for the evaluation and has the great merit of presenting in analytical detail the elements in favor and against any conclusion. This is a great merit as the process from scientific evidence to public policy is too often 'unintelligible' (see for instance the absence on information regarding EEC evaluations on toxic substances). The process adopted is most useful when evaluating items with uncertain scientific evidence.

On the other side the document provides little information to the reader on the scientific information that was considered and the reader needs to go to the original papers. This may be disappointing but avoids the subjectivity inherent in the process of summarizing a paper in a few statements.

The process of evaluation was quite long and, unfortunately, started before the publication of the paper by Ahlbom et al [1] on childhood leukemia. Another paper that was missed but is less important is that by Schuz et al [2].

My comments will focus mainly on the issues related to childhood neoplasm and to leukemia as well as on the general parts (executive summary, introduction, conclusion).

Detailed comments

Executive summary.

P1 / statement for the general public. I understand (not at the first reading) that all 3 reviewers agree on statements from line 7 to line 14 while statement in lines 15-16 indicates some disagreement. If so, this may be made clearer.

P3, line 16: electrical sensitivity is a very unclear concept. I would prefer to discuss it separately from the other diseases.

P4 / lines 18-19 It becomes clear to the reader that reads the entire document that the disagreement with NIESH (and I guess with the more recent IARC) evaluation depends on the different weighting of the information rather to new information. Is it worth to state this more explicitly here?

P4 / lines 21-27. The statement on the potential population burden is based on very uncertain information and will be probably interpreted very differently according to the 'a priori' of the reader. However I have no suggestions to improve its expression.

P 5 / lines 4-7 I would add here that the diseases under discussion are very rare and therefore also the baseline lifelong cumulative incidence is low.

P7 / lines 18-20 I strongly agree with this statement and with the following on the biological mechanism.

P8 / on the contrary, I am confused by the calculations in line 57 and I am not convinced that the number of extra cases can be computed on the basis of the uncertainty of our evaluation.

P9 / table 1 (and in other parts of the document): the terms used are not always standardized, for instance it would be convenient to use the IARC nomenclature in the column IARC Class.

Pages 14-15 The description of reviewers' processes of evaluation is rather complex. This is not a point but while reading this page I questioned myself about the attitude of an 'average reader'.

Page 15 line 23. I do not understand 'visibility'.

Page 16, point 9 I return to my previous comment about the inclusion of Ahlbom's [1] study. Besides the update of references, it is useful in defining the threshold for childhood leukemia.

Page 17 / table 10.1 In the line 'Miscarriage', would it be possible to give the added risk per pregnancy? (I assume it is very close to the annual risk). It would be possible also to estimate a lifetime risk on the basis of the average number of pregnancies, and assuming that the risk does not vary with the number of pregnancies.

In the line child leukemia I would add the age range (0-14 or 0-19?).

Introduction.
I do not repeat the comments anticipated in the comments on the executive summary. In general concepts are more clear here, probably because space constraints are more relaxed. I read with interest the description of the assessment procedure and of the application of Bayesian methods. It is a quite clear description. Percent intervals in page 23 overlap. From a purely formal point wouldn't be better to define non-overlapping intervals?

I focus my comments on the critical side more than on the positive comments. Given this general warning, I would like to express my appreciation for some clear statements on common questions, such as the one on time trends (p. 25 and following). In this respect it may be useful also to consider the evidence of an increase of incidence of lymphatic leukemia in childhood and the progressive appearance of the peak in age 1-4 (see a recent paper from Milham). This evidence is no proof of an EFL effect but it is compatible with it (and with other causes, of course).

Who stated the arguments in pages 27-28? Is it an exercise done by the evaluators team or was it based on external inputs?

P 30, l 10-12: the statements include precise figures on the probability of RR=1. On what distribution are based?

p. 31, l 15: a typing error "..if any. Should" makes the statement hard to understand. Other typing errors appear in the text but usually do not interfere with comprehension.

I would state somewhere that 1 mG=0.1 microT.

It may be interesting to note that the distribution of TWA exposure in table 3.1.2 and figure 3.1.1 is similar to the results we observed in Italy during the pilot study done for our case control. I will send in a separate e-mail a draft (the corresponding paper was published in the proceedings of a congress in Greece in October 2000. All details are in the other mail).

Symbols in table 3.1.1 are corrupted on my hard copy. What do they mean?

The section on animal studies is quite detailed and I do not have any point comments. I have only a general point: In your discussion about animal studies you stress the difference between 'animal' and 'human ' exposure. I wonder if other arguments could be discussed also:

- no animal studies were designed to address an outcome such as infant leukemia (I wonder if any studies on this outcome would be possible);
- how many lab studies are designed to address RR lower than 2?
- The assumption of increasing effect with increasing dose is estimated from studies on agents that damage DNA, therefore may not apply to ELF.

Page 70, line 57: you consider age 20 as the limit for childhood cancer. I would also estimate figures for age 0-10 or 0-14 since the results are actually bases on studies including this age range.

On time to time the statements used are slightly different (e.g. p 72, line 14: 10 to 50% likely vs. 50 to 90% possible) are these real differences? I recommend to be very consistent in order to put the reader in a better condition when reading the document.

Leukemias

p.73, fig. 8.1.1 There are two papers by Kheifets published in 1997 in the references.

Id. fig 8.1.2 how are the studies sorted?

See my previous comments on the paper by Ahlbom et al (2000).

In table 8.1.3 I would add the numbers of controls. I would also indicate separately the results based on 24-48 hours measurements.

Table 8.1.4 I would separate the studies according to the studied groups (for instance: electrical + telephone wks, cohorts or cc in ad hoc studies; id. using census data; results obtained from studies designed for other purposes, such as cohorts of welders; residential studies etc.).
The table includes only a few studies on residential exposure: what are the criteria of selection? I would prefer a full list in a separate table.

Table 8.1.5: I would add the number of subjects. One frequently raised comment is that the number of subjects in the highest exposure category is very low and that results may be due to selection bias (see Kleinerman et al [3]). I am not convinced of that explanation since too many studies of different design and in different areas show RR> 1 and moreover the Scandinavian studies are not affected by such a bias. I would discuss this possible bias more explicitly.
The comment in page 90 on Preston-Martin's study is very important in my opinion. Is it possible to show the figures?

I would split tables 8.2 (arguments for and against causality for leukemia) in one set focused on childhood leukemias and one on adult. For instance the argument A1 in page 93 applies for adults but not for children.

Page 91 Did you include viral infections (a suspected risk factor) among the possible confounders?

Page 94 Argument F1: the same conclusion was reached by the meta-analyses conducted by Wartenberg et al [4]and by Loomis et al. [5]. Is it worth quoting also them?

Regarding Train conductors, their exposure is to frequency 16 Hz.

Page 95. Argument A1: Actually most studies could not investigate this issue appropriately because of limits in their size.

Table 8.2.8: the arguments are not very clear.

p.100 and following: I would discuss in more details the possible selection bias (see previous comment).
Id. line 64: Greenland's study is unable to exclude the absence of a dose response trend, although its point result does indicate it.

Page 108 Milham's study is quoted as "…child leukemia rates ….".

I agree that new studies are not expected to change main conclusions but the data base is still limited in the category with the highest exposure.

Brain cancer:

P. 137, c2 : the statement is not clear. What studies are considered in this statement?

I have no more comments on this point.

Reference List

1. Ahlbom A, Day N, Feychting M, et al: A pooled analysis of magnetic fields and childhood leukaemia. Br J Cancer 2000; 83: 692-698.

2. Schuz J, Grigat JP, Brinkmann K, Michaelis J: Residential magnetic fields as a risk factor for childhood acute leukaemia: results from a German population-based case-control study. Int J Cancer 2001; 91: 728-735.

3. Kleinerman RA, Kaune WT, Hatch EE, et al: Are children living near high-voltage power lines at increased risk of acute lymphoblastic leukemia? Am J Epidemiol 2000; 151: 512-515.

4. Wartenberg D: Residential magnetic fields and childhood leukemia: a meta-analysis. Am J Public Health 1998; 88: 1787-1794.

5. Loomis D, Lagorio S, Salvan A, Comba P: Update of evidence on the association of childhood leukemia and 50/60 Hz magnetic field exposure. J Expo Anal Environ Epidemiol 1999; 9: 99-105.

Comments on Draft 3 of California EMF Program Document:

"An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations, and Appliances"

Submitted to:

California Electric and Magnetic Fields Program
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Attention: Dr. Raymond Richard Neutra

Submitted by:

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September 10, 2001

Statement of Qualifications of Reviewer

David L. McCormick, Ph.D., D.A.B.T. is Vice-President and Director of the Life Sciences Operation at IIT Research Institute (IITRI), Chicago, Illinois. Dr. McCormick also holds the rank of tenured Professor at the Illinois Institute of Technology (IIT), where he teaches graduate courses in toxicology, and graduate and undergraduate courses in physiology. He joined the IITRI staff in 1979, and the IIT faculty in 1982.

Dr. McCormick received his A.B. degree in 1974 from Middlebury College, Middlebury, Vermont, with a joint major in chemistry and biology. He received his M.S. (1976) and Ph.D. (1979) degrees in Environmental Medicine/Biology from New York University (New York, New York). Dr. McCormick received his board certification (Diplomate status) in toxicology from the American Board of Toxicology, Inc., in 1983, and was recertified in 1988, 1993, and 1998.

Dr. McCormick's primary research activities are in the areas of carcinogenesis and cancer prevention, preclinical and environmental toxicology, and the biological effects of magnetic fields. He has published more than 175 research papers, abstracts, and reviews in these areas. He currently serves as Principal Investigator on 11 research programs and 5 Master Agreements supported by the National Cancer Institute (NCI), and has conducted numerous other grant- and contract-supported programs for the NCI, the National Institute of Environmental Health Sciences (NIEHS), and commercial sponsors.

Between 1992 and 1998, Dr. McCormick served as Principal Investigator for the NIEHS/National Toxicology Program project entitled "Studies to Evaluate the Toxicity and Potential Oncogenicity of 60 Hz Magnetic Fields in Laboratory Animals." This program is widely considered to be the most comprehensive evaluation of the biological effects of power frequency magnetic fields ever conducted in experimental model systems. He has also served as Principal Investigator on two NIEHS grants in the field of bioelectromagnetics, and has served as a technical advisor to three additional bioelectromagnetics grants conducted by scientists working in his research group.

Dr. McCormick has served on several dozen grant and contract review committees for the NCI and other funding agencies, and from 1996 to 2000 served as a member of the National Institutes of Health Reviewers Reserve. He is also a regular reviewer of manuscripts submitted for publication in *Cancer Research, Carcinogenesis, Nutrition and Cancer, Cancer Letters, Radiation Research,* and *Bioelectromagnetics,* among other journals. In June, 2001, Dr. McCormick served as an invited participant in the International Agency for Research on Cancer (IARC) meeting to review and evaluate the possible risks of human cancer resulting from exposure to ELF electromagnetic fields.

Dr. McCormick is a member of the American Association for Cancer Research, the Society of Toxicology, the Society of Toxicology Carcinogenesis Specialty Section, the Society of Toxicology Midwest Regional Chapter, the Society for Experimental Biology and Medicine, the International Association for Breast Cancer Research, and the American Association for the Advancement of Science. He has served on the Boards of Directors of the Illinois Cancer Center, the Society of Toxicology (Carcinogenesis Specialty Section), and the Society of Toxicology (Midwest Regional Chapter).

Responses to General Questions

1. Several physicists have made the theoretical argument that the energy contained in 50/60 Hz magnetic fields is insufficient to induce biological effects of any type. Although this argument is attractive in its simplicity and absolute nature, it is not supported by empirical evidence gained from studies of signal amplification in well-studied biological systems. Perhaps the best example of this process occurs in the vertebrate retina. Experimental studies have demonstrated that one photon of red light contains approximately 3×10^{-19} joules of radiant energy. Capture of a single photon of light by a vertebrate photoreceptor cell produces a receptor current of approximately 5×10^{-14} joules; thus, the energy contained in the visible light has been amplified by a factor of more than 10^5 by the biological system. Should a comparable amplification process be involved in EMF reception by the cell, the energy delivered to a biological system as a result of EMF exposure could greatly exceed the amount of energy contained in the incoming stimulus.

   It is important to note that our understanding of sensory receptor systems provides a **possible** mechanism through which the energy contained in power frequency EMF may be amplified to a level at which biological effects may occur. However, no such amplification mechanism for EMF has been identified. As such, any discussion of possible amplification mechanisms should not be interpreted to support the contention that EMF exposure has biological effects. Rather, this issue is raised to indicate that the possibility of biological activity cannot be excluded solely on the basis of the physical arguments that have been put forth.

   A second, even more important caveat to this comment is that the induction of biological effects is in no way equivalent to the induction of adverse biological effects. Restated, the fact that biological effects of any chemical or physical agent can occur (or do occur) cannot be construed as evidence of human health risk.

2. I have some difference of opinion with the prior degree of confidence expressed by all three Researchers. Although I may not agree with their conclusions, I do understand and accept the rationale underlying the positions of Researchers Two and Three. However, the rationale underlying the conclusions of Researcher One appears to be less objective. For example, without any relevant supporting data, Researcher One appears to use different criteria to accept or reject the possible existence of certain types of effects; this bias skews this Researcher's prior degree of confidence. For example, Researcher One accepts as highly probable some types of phenomena

that have not been proven to exist (*e.g.,* perturbance of organism function by EMF interference with electrical signals), while he rejects other unproven phenomena that would suggest opposite outcomes (*e.g.,* existence of a repair mechanism). My prior confidence would not enable me to accept either type of phenomenon.

Before entering the EMF research field approximately a decade ago, I held absolutely no preconceived notion as to the magnitude of the hazard (if any) that EMF exposure posed to humans. At that time, EMF epidemiology data were conflicting; high quality animal data in bioelectromagnetics were virtually non-existent; and no experimental or epidemiologic database existed for similar agents. However, even without a known mechanism of action, the ubiquitous nature of human EMF exposure provided a compelling reason to conduct hazard assessments of such exposure. Then and now, I look at EMF hazard assessment as a problem in environmental toxicology whose evaluation must be approached by a "sum of the evidence" approach in which several types of information are integrated and synthesized. Lacking data to support one or more cellular, biochemical, or molecular mechanisms of action, empirical data from experimental and epidemiology studies must be given primacy in efforts to establish and quantify the risks, if any, that may be associated with EMF exposure. Ten years ago, such data were either absent or conflicting; at that time, I considered it nearly impossible to predict possible hazards with any degree of confidence. The relevant database in studies of EMF health effects is much more robust today, and supports a more effective evaluation of the possible hazards of EMF exposure.

3. In the general case, the lack of a plausible biological mechanism is clearly not sufficient to discount a consistent pattern of epidemiologic and/or experimental findings of biological effects. It is important to note, however, that such findings would very clearly be strengthened by the identification of an underlying mechanism.

What is absent in the present case is a consistent pattern of epidemiologic or experimental data that support the existence of a significant hazard of EMF exposure. In the case of EMF (and other agents for which the epidemiology database is inconsistent or equivocal), the absence of a plausible biological mechanism does indeed weaken the strength of the argument. Furthermore, the absence of confirming *in vivo* data from animal model systems also undermines the strength of arguments that are based on epidemiology alone. Absent either a plausible biological mechanism or supporting experimental data, my confidence in the modest effects identified in most EMF epidemiology studies is decreased.

4. In my view, and in the view of the recently convened IARC EMF panel, the animal pathology literature is indeed null. However, the DHS Researchers appear to have accorded the results of animal studies relatively little import: according to the draft report, the DHS Researchers "did not let this pattern of [of experimental animal] evidence pull down our degree of confidence in the epidemiological literature," and "for some of us, it actually increased the degree of confidence somewhat." I find this conclusion to be an unfortunate situation in which epidemiologists focus solely on the data set with which they are most familiar and comfortable, and thereby ignore other evidence that could provide critical support to a hazard assessment.

In cases where no clear pattern of risk (or lack thereof) emerges from epidemiology studies, the results of well-conducted experimental studies can provide important insight into the possible risks of agent exposure. A number of EMF epidemiology studies have identified a positive relationship between EMF exposure and the risk of a specific disease. However, studies in other, often comparable, populations have often not identified any relationship between EMF exposure and disease risk or outcome. As a result, few scientists would conclude that the sum of the EMF epidemiology literature provides a compelling case that EMF is a causal factor in human disease. In my opinion, the lack of experimental data to substantiate the positive findings of some EMF epidemiology should be considered in evaluations of the robustness of those findings.

Although the vast majority of studies conducted in animal model systems have identified no hazards associated with EMF exposure, two types of studies were identified by the DHS as supporting a potential health hazard. For the reasons described, below, the results of these studies should be accorded limited importance.

Studies performed using chick embryos as a model system are not commonly used to identify or evaluate human health hazards. As a result, the ability of such studies to predict human health effects is unknown: essentially no data exist to support the reliability and predictive nature of the chick embryo as an experimental model for human toxicity, and risk assessors and regulators do not commonly use chick embryo studies in developing human hazard assessments.

The significant limitations of the rat mammary carcinogenesis data generated in both the Löscher laboratory and in Soviet Georgia suggest that the reported positive findings from these laboratories also merit only limited consideration. The value of the Georgian studies is limited by inadequate description of EMF exposure methods and monitoring, and by other concerns related to experimental conduct. As discussed in a previous peer-reviewed publication (Boorman *et al.,* Magnetic Fields and Mammary Cancer in Rodents: A Critical Review and Evaluation of

Published Literature. **Radiat. Res. 153,** 617-626, 2000), the results of Löscher and colleagues are inconsistent between experiments: various papers from this laboratory report increases in mammary tumor incidence, mammary tumor multiplicity (but not incidence), or mammary tumor size (but not incidence or multiplicity).

Of even greater concern is the fact that the dose-response parameters for mammary tumor induction by DMBA that have been generated in the Löscher laboratory differ from those that have been published by nearly every other laboratory in the world (including my own laboratory, and the Anderson laboratory at Battelle-Pacific Northwest laboratory in which unsuccessful attempts were made to replicate the Löscher work). Prior to the publication of their first EMF study, the Löscher laboratory had no peer-reviewed publications in rat mammary carcinogenesis, and no other record of experience using this model system. This lack of experience is troubling, particularly when considered with the fact that the DMBA dose-response data generated in the Löscher laboratory do not agree with results generated in more than twenty laboratories over more than three decades. I must presume that the EMF community is not sufficiently familiar with the literature in rat mammary carcinogenesis to be aware of this critical discrepancy. Yet, the work was identified in the DHS report as a key "hypothesis generating" study, and appears to be considered by some in the EMF community as the "gold standard" for breast cancer studies. The quality of the Löscher work clearly does not merit such standing.

5. In the abstract mathematical sense, there is little doubt that a relative risk that is calculated to be between 1 and 2 may be real. Assuming that such an increase in relative risk is indeed real, the more important issues are (a) whether such a small increase in risk can be demonstrated with any degree of statistical certainty, and (b) whether an increase in risk of that magnitude is important to public health. In answer to part (b), it should be clear that in situations where even a modest elevation in risk is associated with broad population exposure, this small increase in relative risk could be an important determinant of disease incidence.

However, demonstrating that such a small increase in risk is indeed real (and is not a function of confounding, bias, or random behavior within the study population) presents major challenges to study design and analysis. In this context, it is important to consider the limitations of epidemiology: whereas epidemiology is very good at identifying rare events superimposed on a background that is near zero (*e.g.,* angiosarcoma of the liver in workers exposed to vinyl chloride monomer) and high incidence events (*e.g.*, lung cancer in cigarette smokers), it is much

less powerful at identifying small increases in incidence that are superimposed on a non-zero background. I will reiterate that in cases where the epidemiologic data are equivocal or conflicting, consideration of the results of studies conducted in experimental animals can be essential to the development of a hazard assessment. The results of the large body of high quality experimental studies that were designed to evaluate the risks of EMF exposure appear to have been accorded only limited importance by the DHS Researchers.

6. I would propose that lack of specificity with respect to disease site should most definitely decrease, rather than increase, the confidence that the DHS Researchers place in any observed epidemiologic associations between EMF exposure and cancer risk. It is unclear to me why this lack of specificity should increase anyone's level of confidence in the findings.

Cancer is a family of diseases, rather than a single disease entity. Although neoplasms arising in different sites often demonstrate important similarities, different molecular alterations appear to underlie neoplastic transformation in different tissues. Furthermore, the kinetics, types of growth, responses to pharmacologic intervention, and other biological parameters often demonstrate a wide range of differences between sites. It is clear from both human and animal data that environmental agents demonstrate organ specificity in cancer induction; what is not clear from the cover letter to the Report is why our growing understanding of cancer biology and differences among tumor types should be not be integrated into EMF hazard assessments.

7. Specific comments are made in response to individual arguments.

8. Suggested alternative terms are provided in the Table below.

Confidence Range	Current Phrase	Suggested Alternative
>98%	Virtually certain	Virtually certain causality
90-98%	Highly probable [sic]	Highly probable
50-90%	Possible >50%	Probable (≥50%)
10-50%	Possible <51%	Improbable (<50%)
2-10%	Very improbable	Highly Improbable
<2%	Virtually certain that it is not causal	Virtually certain lack of causality

Comments on Specific Content of Elements of Tables

Page #	Table #	Line # or Comment # in Table	Comments
41	6.1.2.		Table is missing several relevant papers (all demonstrating no increase in leukemia/lymphoma incidence in either standard or transgenic mice exposed to EMF)
42	6.1.3		This table involves studies of skin tumor promotion, whose primary product is a squamous cell papilloma (a benign lesion). As such, these studies do not involve skin cancer. Table is missing several relevant papers (some demonstrating co-promotion by EMF; most demonstrating no effect of EMF on skin tumor promotion or co-promotion)
43	6.1.5.		Table is missing papers from Yasui *et al.*, and Mandeville *et al*., both of which found no effects of EMF in two-year bioassays conducted in rats
43	6.1.6.		Table is missing a very large number of relevant papers reporting teratology/reproductive toxicology studies in standard animal model systems; all papers reported no effects of EMF exposure
44	6.1.7		Table is missing paper (Boorman *et al*, 1997) presenting results of 8-week NTP-EMF toxicology studies (including hematology and clinical chemistry
45	6.1.8.		Table is missing several papers, including the most comprehensive immunotox bioassay of EMF (House *et al.*, 1996), and a replicate study of NK cell function (House and McCormick, 2000).
56	6.2.1.	F1	The data from Löscher's group are NOT consistent across studies. In some studies, increases in tumor incidence were found; in others, no increase in incidence but an increase in multiplicity was found; in yet others, no changes in incidence or multiplicity were found, but an increase in size was reported. Some of the reported findings are well within the variability of the model system.
56	6.2.1.	F2	The high mammary tumor incidence seen in Anderson's attempts to replicate the Löscher data are consistent with the dose-response data from virtually every laboratory in the world. It is the Löscher data, not the replication data, that is the outlier. Furthermore, how robust (and of what significance) is a finding if it cannot be replicated with minor changes in study design?
56	6.2.1.	C1	The "hypothesis-generating" study yielded DMBA dose-response data that are inconsistent with the results of studies conducted in numerous other laboratories over more than 30 years. The Löscher data appear to be fundamentally flawed.
57	6.2.2	F1, F2, F3	How can it be stated that these arguments support causality? They absolutely do not. There is not doubt that these arguments identify important (and valid) limitations to the results of animal studies.

			However, they do not provide any evidence to support a causal role for EMF in leukemia or lymphoma induction.
57	6.2.2.	C1	It is impossible to prove a negative. The lack of a positive finding must be considered as such, in consideration of the limitations of the experimental designs.
57	6.2.2.	C3	This statement is simply incorrect, and suggests a total lack of understanding of both chronic oncogenicity evaluations and carcinogenic mechanisms. Chronic animal exposure studies can (and do) identify non-genotoxic carcinogens. This statement also suggests that the entire animal experimental database for EMF and leukemia/lymphoma was not considered. Several studies in transgenic animals were designed to identify possible promoting effects in mice that are genetically predisposed to lymphoma and/or received prior exposure to a chemical carcinogen. These studies were all negative.
57	6.2.3		All of these studies evaluated effects on the induction of squamous cell papillomas; these are benign lesions, not cancers.
57	6.2.3.	C3	Virtually all studies were conducted to identify effects of EMF as a promoter or co-promoter. This statement is not appropriate for the discussion of skin tumorigenesis.
58	6.2.4	F2	How does this represent an argument supporting causality? Statement F2 is a valid statement of a limitation of experimental animal data, but it does not provide evidence supporting causality.
58	6.2.4	F3, C3	Chronic animal exposure studies can (and do) identify non-genotoxic carcinogens; these agents would not act through an "initiator" type of mechanism. This statement is simply incorrect.
58	6.2.5	C3	Statement is not appropriate for this model system; both reported studies involved multi-stage ('initiation-promotion") designs.
59	6.2.6.	F2 – F5	The strength of the positive chicken findings is GREATLY outweighed by the large number of negative studies conducted in well-studied rodent models with demonstrated predictive ability for humans.
59	6.2.6	F3, C1	Discussion of theoretical models is inappropriate for this section – as stated above, the presence of "biological effects" is not the same as the presence of adverse effects.
59	6.2.6	C3	The overwhelming number of negative rodent studies swamps the limited evidence generated in chickens. In consideration of both the volume of data and our understanding of the relative predictive nature of the models, the rodent data should be accorded much more weight than the chicken data.
59	6.2.7	F1	The largest and most comprehensive studies (NTP, Mandeville, and Yasui, none of which was included in the summary) are uniformly negative. These findings greatly outweigh the results of more limited endpoint analyses.
60	6.2.8.	F1	The most comprehensive studies (House) were not included in the analysis.

61	6.2.12	A1	The recent consensus among researchers in the melatonin field (working in both animal and human systems) is that EMF has no effect on melatonin synthesis, secretion, or levels
61	6.2.12	F1	Some laboratories (*e.g.,* Reiter), who previously reported positive effects, no longer find effects after replacement of their exposure equipment with more instruments that are more carefully designed, controlled, and monitored. The bulk of the recent evidence is that EMF has no effect on melatonin levels.
64		6-36	The huge emphasis on the henhouse results is curious, if not absurd. The chicken reproduction model used is of minimal relevance to human reproduction, and the data from the henhouse studies are clearly outweighed by the results of a large body of reproduction studies in rodent models whose relevance to human health is understood.
64		48-49	The fact that the Löscher dose-response data for DMBA does not agree with the data generated in numerous laboratories over three decades greatly undermines the credibility of his results with EMF. These studies should be deemphasized.
64		57-59	On what basis can the statement be made that false negatives are more likely than false positives? It is clearly impossible to prove a negative; however, one can establish limits for an effect that can be excluded through such a negative result. Absent ANY clear evidence of a reproducible, robust EMF effect in animals, this last paragraph is very difficult to accept.

The views expressed in this document are solely the author's and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas & Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District, and San Diego Gas & Electric Company.

To: Raymond Neutra
California Department of Health Services

As you requested, I am sending you comments on the third draft of the DHS Electric and Magnetic Field (EMF) Risk Evaluation. For each of the questions posed, the approach I have taken in providing revised comments is to consider how I responded to the issue in my comments on the second draft and how the issue has been addressed in the third draft. These revised comments are consistent with my verbal comments at the August 7, 2001 meeting of the EMF Science Advisory Panel.

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1) *…We say that theories should be used to predict results that are falsifiable not used to discount evidence. Thus, our prior degree of confidence is not vanishingly small. How sound or flawed is our discussion and this position for a risk evaluation.*

In my comments on draft two I supported this perspective and expressed the view that the discussion is sound and provides adequate justification for the position. My earlier comments still apply to draft thee.

2) *Each reviewer was close to 90% confident a priori that easily detectable relative risks would not be produced...*

As was my comment on draft two, I have found that in draft three, the reasons for these judgments seem reasonable. Draft three has addressed my comment that draft two did not have a clear statement explaining whether the experts did or did not confer among themselves.

3) *We were not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects and then a chain of events that led to pathology. Yet we did not let this pull our degree of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment.*

As noted in my comments on draft 2, I find this stance reasonable.

4) *We viewed the animal pathology literature as largely null with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment.*

In my comments on draft 2, I expressed the view that stance is reasonable and I sill hold this opinion.

5) *Not all epidemiologists would agree with our position that relative risks between 1 and 2 should be taken seriously unless there is specified evidence for confounding or bias to explain it away. Are we justified in taking that stance?*

I have no comment on this issue

6) *We said that a lack of specificity in the association of EMFs with subtypes of cancer and evidence for effects on various types of disease did not pull down our degree of confidence and might even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature. Do you agree? Please comment.*

In draft two, I found your stance reasonable and gave examples to support my finding. With regard to draft 3, I believe the stance is still reasonable.

7) *Have we done an adequate job in presenting the arguments for and against causality or are we assigning weak arguments to the con or pro position.*

The arguments for and against causality appear to be balanced. I did not get a sense that there was a systematic effort to assign weak or strong arguments to one or another side of the issue.

8) *Our Risk Evaluation Guidelines (REGs) define some "plain language phrases" to express our degrees of confidence. However, when we actually applied them we found they were not problem free:*
   a) Some of these phrases are not mutually exclusive. For example,
   Possible >50% overlaps "highly probable" and virtually certain."
   "Possible<51%" overlaps "Possible >50%". In this case, the overlap is slight,
   but important, since it is about the "balance of probability".
   b) These phrases are grammatically awkward and they are not really "user friendly". How could we rephrase them, without violating the spirit of the REGs? Please write any suggestions next to each phrase:

Confidence range	Current Phrase	Suggested alternative
>98%	Virtually certain	***Virtually certain***
90-98%	Highly probably	*Highly likely*
50-90%	Possible >50%	*More likely than not*
10-50%	Possible <51%	*Possible*
2-10%	Very improbable	*Very unlikely*
<2%	Virtually certain that it is not causal	Virtually certain that it is not causal

Department of Health Service
Prof. Raymond Neutra
Division of environmental and occupational
Disease control
1515 Clay Street, Suite 1701
Oakland, CA 94612

Tel.-Durchwahl 17-3252 Datum 19.09.01

EMF risk evaluation

Dear Raymond,

In the meantime I found some time to look into your report, although I must admit that I have no chance to read everything in full detail !

At first I want to congratulate you and your co-workers for the large amount of work which you have invested in preparing the report. The different evaluation perspectives which you also presented in Garmisch are very interesting and illustrative.

Coming from our own work in the field I would like to comment mainly on the issue of childhood leukaemia.

At first a technical remark: Due to the time of finalising your report you quote in table 8.4.9 our study published earlier this year amongst “studies in pipeline”. I think that this is inevitable if you don’t want to rewrite the report each time a new publication has appeared. Therefore you might only correct the quoted sample size (514 cases and 1301 controls instead of 200 each):

With respect to your arguments for causation I have the following remarks:

- Depending on how exposition is measured misclassification may also lead to falsely increased risk estimates (but wider confidence limits).
- Selection bias: Nearly all studies are biased in the form that
    - Participation rates are greater for cases than for controls
    - Controls tend to have higher SES
    - Higher SES is associated with lower exposure to residential EMF

  These points may lead to a spurious association due to selective participation. According analyses have been published by Hatch et al. and have also been performed in our study by Joachim Schüz. However, selection bias does only explain parts of the observed associations

- The argument of consistency among the cited 19 studies holds only partially: In many studies several methods of exposure assessment have been applied and in some instances the authors seem to have picked the results for publication which showed the greatest risk estimates. Thus the apparent consistency – and also some results of meta-analyses – are based on criteria which differ quite substantially.
- The estimation of the attributable risk for childhood cancer appears to be somewhat high in some of your scenarios.

I am sorry that I only had the time to send these few comments. However, I hope that they may be at least of little use for your group.

With best regards

Yours sincerely

Joerg

Dear reviewers,

I think that you are to be congratulated for doing a first class job on what I consider to be a thankless task. I have no major problems with what you did, how you did it, or with your conclusions. I'll respond to the numbered questions in the order they were asked.

1. I agree. Theories which arise can often be tested with available data. Epidemiologic evidence should stand until refuted by better studies.

2. One dramatic change in disease rates after the introduction of electricity was the emergence of the mortality peak at age 4 of childhood leukemia. This has been shown to consist solely of common acute lymphoblastic leukemia. In the US, the urban to rural spread of electricity between 1920 and 1955 is strongly correllated state by state with residential electrification. I'm convinced that this type of leukemia is nearly completely attributable to EMF's. (see Milham and Ossiander, Med. Hypotheses (2001) 56(3),290-295.

3. I agree. My favorite mechanistic theory has yet to be tested. There is some evidence that EMF's cause a failure of some parts of the immune system (in the U of W germ free rat study (Kunz et al, and in my study of healthy aluminum workers (Davis and Milham . (1990) AJIM 18, 79-85, immune cell phenotypes were similarly altered). Immune system failure could explain much of the EMF related pathology.

4. I agree. However, I think that the animal exposure studies done with other parts of the EMF spectrum should also be considered. The U of W rat study showed a large cancer excess in the microwave-exposed rats (18/100 cases vs 4/100 controls).

5. I agree. Most of the epi studies are of the case-control type, with an absense of clean controls. This gives low risks. With the fact that the entire population is exposed to EMF's, a small risk increase will effect large numbers of people.

6. I agree. In my studies, there is evidence that some of the adult leukemia subtypes have an increased risk.

7. An adequate job.

8. There is a typo in the current phrase for the 90-98% confidence range. It should be highly probable, not highly probably. Any arbitrary ranking scheme will have problems. this one is as good as any.

In table 1of the executive summary, I was surpised that there was such a difference between reviewers' degree of confidence in causality in childhood leukemia. I'm in agreement with reviewer no. 1.

In table 2 I think that about 70% of childhood leukemia deaths will be attributable to EMF's

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From: Susan Molloy [mailto:susanm@cybertrails.com]
Sent: Thursday, August 09, 2001 9:02 PM
To: Neutra, Raymond (DHS-DEODC)
Subject: OK, here are my real comments, to include.

Dear Dr. Neutra,
I read the Risk Evaluation report produced by the California EMF Program and very much appreciate that CDHS is beginning to examine em field issues.

For personal reasons, I studied especially Appendix Four, "Study Review of Hypersensitivity of Human Subjects to Environmental Electric and Magnetic Field Exposure," hoping for news of progress. The author reported on the Literature, but his experience could do with rounding out. I urge your researchers to please dig deeper into the health issues of people injured or even disabled by common emf exposures. I'm certain the Literature search is useful in many respects, but it missed the mark, at least for me - the "mark" being that many people truly are being made extremely ill by exposure to certain common fields, and the risks that we face have gone unaddressed in the report.

Some live with reactions such as chronic headaches from cellphones or fluorescent lighting, while others of us have had to move away from the settled areas, where we would very much prefer to be, on the grid in order to be able to take care of ourselves and our responsibilities, and to stay out of nursing homes.

How can it be possible that there is no Literature about all the people who landed at the wrong end of the bell curve? If this is the case, please come meet us where we are, and create the Literature. There has to be a way I believe you can benefit by learning about the condition, often linked to emf reactions, called hyperacusis. It means a person can "hear" or sense electrical equipment, fluorescent ballasts, VDTs, rechargers, etc., which trigger nausea and for some of us, disturbing neurological reactions.

For me, some combination of emf susceptibility and hyperacusis results in immediate loss of coordination and orientation, and triggers movement disorders (dystonia, choreoathetoid movement). My speech and gait are affected with first a surge, then become worn out. I've had complete loss of voluntary motor control to the point of temporary paralysis. All I can find to read about things like this abstracts from weaponry researchers at Oak Ridge Laboratory. They are studying how to drop enemy troops or civilian demonstrators using aimed em fields, with no bloodshed. I experience other self-destructive repetitive behavior such as interrupting people, and loss of adult social graces and impulse control, as well.

I lose the sense of how light or heavy I am on the earth. If near a large emf source like Bay Area Rapid Transit or laundry equipment, or certain noises like a fire alarm, a vacuum cleaner, or the Blue Angels flying overhead, I've been dropped to the ground.

The closest descriptions I can find about what this feels like are in the end section of Oliver Sachs' book "Awakenings." I figure, the emfs and sound combination might impact people with Parkinsons Disease, who (if they can still communicate) refer to "Saturnian Gravity," feeling so heavy they cannot lift their arms or heads, or take a step. If their heads are tilted backward, and they're in their wheelchairs, their eyes can roll upward and get stuck. This happens to me with bad exposures, and it is very annoying. When I go into cities, airplanes, or most large public places for even a little while, my coordination is impacted and I frequently have to be hauled around in my wheelchair which is a big bother.

I don't know who to talk with about how these symptoms can be triggered so rapidly upon exposure, and how then I recover, typically by the next day. The reactions are exhausting and usually I go to sleep, then wake up groggy and have to become reoriented.

These episodes are not epilepsy, at least not any kind of which I'm aware. I can still hear people's voices, although it seems like I am listening almost from underwater. It doesn't make sense that a person could have Parkinson's

Disease, Williams Syndrome, Dystonia, or another chronic or genetic condition with symptoms like this for just two hours, or for a week, then recover after being taken away from the exposure and resting.

I figure there's some touchy, vulnerable part in my head that either swells up, has scar tissue on it, or some other small ding so when I'm exposed to the emfs and motor noises there's a rapid irritation or swelling, or interruption of an enzyme or a neurotransmitter, or some other tiny but critical fluid or mineral.

I'm an adult and it's my responsibility not to put myself in harm's way, or else to go ahead and take the "hit" when that's my best choice, for something that I know is worthwhile. However, there are a lot of little kids who may be having hyperactivity, impulse control trouble, no memory for their schoolwork, or have related problems who are exposed to computers, fluorescents, the aerated fishtank in their classroom, their own cell phones, or other nonessential motors. I'm afraid that they are unwittingly being set up for neurological chaos, like my own, later in their lives.

I was helped with focus and became less minutious for a month and 1/2 by taking Dexedrine prescibed by my doctor, evidently the result that's sought for ADHD children. It beat the symptoms back enough that I learned how to do tasks on this computer, such as reading your report and sending e-mail. (It is custom metal-shielded, and the display is a vertical 3-M projector screen, lit by five 25-watt light bulbs, instead of a VDT or LCD. The computer itself is remoted into a far room, in a metal clad box, and the well-twisted wiring runs through conduit above the ceiling.)

After those few weeks on Dexedrine, the escalating side effects and increasing agitation made it impossible to take any longer. I stay in therapy to help with job stress, and my own self-consciousness concerning acting out, plus having turned out to be so different than I'd expected. My current meds are helpful and are carefully monitored, and I choose which risks to take thoughtfully - I do truly have a sense of "spending" my life. It works out.

I don't expect you to know at this point how to help me or the others like me, and I do realize how strange it all may sound to people from the worlds of research and engineering.

However self-conscious it makes me to have written you these comments, I felt a responsibility to mention that while your work on the Risk Evaluation is appreciated, there are some extremely critical issues left not finished. For some purposes, we are only at the extreme beginning. I think you already know. Godspeed.

Sincerely yours,

Susan Molloy

August 31, 2001

Dr. Raymond R. Neutra
Electric and Magnetic Fields Project
1515 Clay Street, Suite 1700
Oakland, CA 94612

Re: "An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances"
Draft 3, April 2001

Dear Raymond:

Below I provide my comments on the above EMF report in two sections. First, I will give you my unstructured reactions to the report (which are not necessarily presented in order of importance). Although I think the report has a number of positive attributes and strengths, I will focus on my perceptions of problems, questions, and the need for change. Except where noted, my comments do not refer to specific lines in the report but are more general in nature. In fact, I raised most of these points at the August 7, 2001 meeting of the Scientific Advisor Panel. Second, I will respond briefly to each of the 8 questions in your letter of July 9, 2001.

Reactions to the Report

a) Although the original idea for this report included a formal Bayesian analysis of the epidemiologic evidence, that approach was abandoned in favor of an informal Bayesian-like approach in which each of the three expert reviewers subjectively weighed the evidence and generated posterior probabilities of causation for each outcome. I am concerned that modification of one's priors in this way may not be very reliable and that certain methodologic issues were not adequately taken into consideration (see below for elaboration). Thus, I am not convinced that abandoning a formal Bayesian analysis was justified, and I do not find the rationale for this shift very compelling. On page 22 (lines 53-57), the report states, "A full probabilistic Bayesian assessment...is so dependent on what can only be educated estimates of the prior for disease effect and for sources of bias and confounding, that it requires complicated and probably unintelligible modeling and cannot be done with a simple calculator and the Bayes equation above." I do not appreciate the relevance of this statement about priors because such priors are also required in the subjective approach used in this report, and I am not convinced that the probabilistic treatment of possible biases would produce "unintelligible" results. On page 71 (lines 51-53), the report maintains that "our stakeholders made clear at the outset that we should not rely on a method that was not be (sic) accessible for criticism to most readers." I do not understand how the results of a formal Bayesian analysis would be less "accessible for criticism" than the results presented in this report. Readers of the report need not understand all details of the statistical methods in order to understand the approach in general and be able to interpret the findings and make criticisms.

b) I do not necessarily disagree with the priors or the evaluation of non-epidemiologic evidence by the three expert reviewers, but it is clear that many other potential reviewers, including certain members of the Scientific Advisory Panel, would have different priors and would give the non-epidemiologic evidence much more weight. I think these other views should have been represented by additional reviewers for this report. This point is especially relevant because the three reviewers worked together, had a similar epidemiologic orientation, and regularly shared their ideas about EMF issues with each other.

c) I believe there are several problems and omissions in the evaluation of epidemiologic evidence addressed in this report (see elaborations below). In general, I think there might have been a tendency with certain outcome conditions to generate posterior probabilities that are too high. In many cases, however, my main point is

not simply that I disagree with the reviewers assessment or posterior probabilities, but that certain methodologic issues were not sufficiently addressed in the report. From our discussions at the last Scientific Advisory Panel meeting, I got the impression that some of these issues were actually considered by the reviewers, but the report does not reflect these considerations.

d) All three reviewers used epidemiologic evidence almost exclusively to change their confidence in causation; and, as shown in Table 8.2.15 for leukemias (p. 99), the primary criteria for changing their confidence in causation were "consistency" (the extent to which all estimated risk/rate ratios [RR] are greater than one or less than one) and "homogeneity" (the extent to which all estimated RRs are the same magnitude) (see definitions on p. 65, Table 7.1). It seems to me, however, that these two criteria are redundant; they involve an evaluation of the same evidence. I am therefore led to believe that the reviewers may have given too much weight to this criterion.

e) A second problem with the assessment of the consistency/homogeneity criterion is that there is almost no discussion of possible "publication bias"–i.e., the tendency for positive findings to get reported and published. Why, for example, is there no mention of publication bias in Table 8.2.5 (p. 93) or in the related discussion?

f) A third problem with the assessment of the consistency/homogeneity criterion is that the reviewers do not make a systematic (quantitative) attempt to examine or explain differences in the estimated EMF effect observed across studies. For example, I could not find any discussion of a meta-analysis of the studies examining the relation between EMF exposure and adult leukemia. (In fact, the RR estimates are not so "homogeneous;" see Table 8.1.1, p. 73). From the results shown in Table 8.1.4 (p. 80), it appears that the estimated RR was lower in cohort studies than in case-control studies. Is that true; and if so, how does it affect our causal inferences? Are there other study-specific factors that explain differences in the estimated EMF effect observed across studies, and what do those findings imply? Similar questions could be asked about other conditions in this report.

g) It seems to me that this report is incomplete and inconsistent about reporting descriptive information for epidemiologic studies of EMF and various conditions. For example, we are not told the design of each reported study of childhood leukemia (see Table 8.1.3, p. 76). How many of these studies were case-control designs?

h) In contrast to the possible over-weighting of the consistency/homogeneity criterion noted above, the consideration of possible "bias" in epidemiologic studies seems to be under-valued or inadequately elaborated. First, in my opinion, two very distinct but important sources of bias have been inappropriately combined into one criterion, called "bias" (see Table 7.1, p. 65): selection bias (which is especially important in case-control and cross-sectional studies); and measurement error/misclassification, especially regarding EMF exposure (which is important in all designs).

i) Second, my impression is that the reviewers used a "non-conservative" approach for evaluating possible biases (selection, misclassification, and confounding). That is, possible biases did not appear to reduce the reviewers' confidence in causation or affect their "degree of uncertainty" (confidence bands) unless there was solid empirical evidence that such biases exist, that their direction was away from the null, and that their magnitude was appreciable. My feeling is that possible biases, even without empirical support, should influence the degree of confidence or uncertainty because they reflect our ignorance. For example, if little is known about the risk factors for childhood leukemia, then possible confounding by unmeasured or unknown risk factors in observational studies should reduce one's confidence in, or increase one's uncertainty about a causal relation between EMF and that condition.

j) Particularly in the case-control studies of childhood leukemia, I am concerned that there is likely to be selection bias that might explain in part the positive findings and that was not adequately addressed in this report (see Table 8.2.2, p. 89). As an epidemiologist who is currently conducting a case-control study of several cancers, I am aware of the difficulty in getting eligible noncases, especially children, to participate as controls, especially in so-

called “population-based” designs in which the controls are supposed to be representative of the population at risk from which the study cases arose. Assuming that eligible noncases are less likely than eligible cases to participate, there will be selection bias if participation is also related to EMF exposure. Specifically, that bias will be positive (i.e., exaggerating the apparent detrimental effect of EMF exposure) if highly exposed children are less likely than minimally exposed children to participate. When I first considered this possible source of bias, I did not think it would be too important because I had no reason to think that participation should be associated with an environmental exposure such as EMF. Now, however, I realize that such an inverse association between EMF exposure and participation might be due to the influence of SES–i.e., high SES children, especially noncases, are more likely to participate and less likely to be highly exposed than are low SES children (e.g., see Hatch et al. *Epidemiology* 2000; 11:189-98). Thus, we would expect positive selection bias, even if SES is not a risk factor for childhood leukemia; furthermore, we cannot control for this bias by adjusting for the effect of SES.

In considering the possible role of various sources of bias in these case-control studies of childhood leukemia, the reviewers should also discuss the results from the case-specular analyses (in which the wire code of each case home is compared with the wire code of a hypothetical home across the street).

k) When evaluating the role of EMF-exposure misclassification in epidemiologic studies, the reviewers seem to assume that such error is non-differential and that the resulting bias will be toward the null (e.g., see Table 8.2.2, p. 89, C2). Although I agree that the direction of this bias is likely to be toward the null, one cannot rule out bias away from the null. Even if EMF-exposure misclassification is unrelated to disease status, it might be related to measured or unmeasured confounders.

l) In their pooled analyses of EMF exposure and childhood leukemia in four case-control studies, Greenland et al. (*Epidemiology* 2000; 11:624-34) found a positive association between wire-code index and childhood leukemia, adjusting for direct individual-level measures of magnetic fields and other potential confounders. What should we infer from this finding? Does it suggest selection bias in the case-control studies? Or perhaps use of the wrong metric in assessing EMF effects?

m) In both the pooled analysis of Greenland et al. (2000) and the meta-analysis of Wartenberg (*AJPH* 1998; 88:1787-94), the authors conclude that there is a need to study more highly exposed populations to assess the possible effect of EMF exposure on childhood leukemia. Do the authors of this report or the three reviewers agree with this recommendation? Why?

n) Although posterior probabilities of causation were expressed graphically in the report as both point estimates of confidence and as confidence bands around each point estimate representing the degree of uncertainty, I saw almost no reference to these confidence bands in the text. Conclusions seem to be made exclusively on the basis of the point estimates. Why? It seems to me that in any Bayesian analysis, even the type used in this report, there should be a clear method for describing the degree of uncertainty and that the conclusions should be based in part on this assessment. Should the reader assume that these confidence bands are uninterpretable?

o) In Chapter 3, I think a better job could be done in describing how one measures the “EMF mixture” and what the units mean (see, e.g., p. 31, lines 24-31). What is the relation between a microtesla and a milliGauss? Is 0.1 μT = 1 mG?

p) It seems to me that the word, “risk,” is used inappropriately throughout this report to mean effect–i.e., the biological effect of EMF exposure on the occurrence of a disease. While I recognize that this use of “risk” may be consistent with common usage among laypersons, in epidemiology, risk refers to the probability that some outcome event (e.g., disease) will occur in a given period–either in a particular person or in a group (average risk).

Response to 8 Questions

1) I agree that the prior should not be "vanishing small," but not necessarily because "theories should be used to predict results that are falsifiable and should not be used to discount evidence." How does one falsify the null hypothesis if that is what the theory predicts? See also (b) above.

2) My priors would be similar to those of Reviewer 2 (see pp. 9, 29-30), and I tend to agree with the reasons given in the report. See also (b) above.

3) I agree that lack of a mechanistic understanding of how EMFs could cause biological effects should not pull down very much our degree of confidence in a (causal) effect. My reason is that historically many associations that were originally regarded as biologically implausible were later found to reflect true effects; moreover, lack of a mechanistic understanding for EMF effects may simply represent our etiologic ignorance.

4) I am not very familiar with the animal literature regarding EMF effects; thus, I do not feel qualified to judge how that evidence would affect my degree of confidence in causation.

5) Yes, I agree that estimated (?) relative risks between 1 and 2 should be taken seriously because: i) they might reflect a large impact on the disease in a population (i.e., attributable fraction) if the exposure is common; ii) they might be biased toward the null; and iii) they might reflect low frequencies of effect modifiers (e.g., due to biological interactions between EMF and other causes of the outcome).

6) I agree that lack of specificity in the association between EMFs and one disease should not reduce our confidence in causation for a given disease. Whether such lack of specificity *increases* our confidence in causation, however, is questionable. First, this increase in confidence depends on the plausibility of multiple or inter-related biological mechanisms. Second, similar associations with different diseases might be due to similar biases.

7) I think one of the shortcomings of this report is that the pro and con arguments for evaluating the epidemiologic evidence are not thoroughly addressed. Refer to my comments above (i.e., c-n).

8) I would label the 50-90% confidence range as "probable" and the 10-90% range as "possible." Do not use probabilities (i.e., >50% and <50%) to further describe each confidence range, which is defined in terms of probabilities.

Sincerely,

Hal Morgenstern, Ph.D.
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The Honorable Hillary Rodham Clinton July 21, 2001
United States Senate
476 Russell Senate Office Building
Washington, DC 20510

Re: EMF RAPID Interagency Committee's Findings
and Conclusions Report dated September 2000

Dear Senator Clinton:

I know from your website that you are actively involved in environmental concerns, some of which are directly related to the subject of possible EMF involvement regarding high numbers of breast cancer cases on Long Island and also in regard to the childhood leukemia cluster at Fallon, Nevada.

I wrote a letter to Senator Paul Wellstone at his Washington office requesting a copy of the above report and also information as to when this report will be presented to Congress. His office advises my request was turned over to the Office of Science and Technology (OSTP). It has been three weeks and I have not had a response.

I am enclosing several documents, a couple of which pertain to the above report and others that provide some background information regarding how I and my family have been affected by two high voltage powerlines that are on the boulevard only 50 feet from the front of our house.

Recently I have recorded readings of over 10 miligauss in our living room and master bedroom. Electrical contractor, Dave Stetzer, from Blair, Wisconsin, who will soon be testifying for Michigan Attorney General Jennifer Granholm regarding power quality problems in Michigan, has recorded transients from the high voltage lines of 120 volts, 220 volts and higher. In one of my enclosures, I mention a 25 volt transient recorded by Dave at my kitchen sink with the POWER OFF!!!!

My enclosures give some background into some of the more serious health problems our family has endured; however, not all of the serious health concerns are mentioned. We constantly worry that two of our grandsons who were diagnosed with rare immune deficiencies may develop leukemia and lymphoma (we were told this may happen). Our daughter just had surgery regarding nodules in her lungs and on her trachea. The 13 nodules in one lung, 8 in the other lung as well as enlarged lymph nodes on her trachea were biopsied (over 20 biopsies) and the results yield only the information that the nodules are "non-caseating granulomas." In other words, "a cause" has not been identified. I have a journal article stating her problem may turn into lymphoma.

You will note also from my enclosures that I make reference to knowing for certain that the blood from my two grandsons with the rare immune deficiencies was sent to an EMF researcher whose work was funded by the National Institute of Health. The immunologist was not willing to share that information with us but had provided the name of the doctor. I found confirmation on the internet.

I provided a limited amount of information regarding our pet guinea pigs and blood test results that indicate a leukemic condition. The Veterinary Clinic at the University of Minnesota opted to tell us "they forgot to do a bone marrow exam." The request was made at time of necropsy of one of the guinea pigs who could not overcome the radiation assault from being on the "powerwall" (where electric meter box is attached to the house), which is the identical situation I discovered my two grandsons were sleeping in (cousins living in homes in different cities).

One of the enclosures (the "Bovine Practitioner" article), cites the exact blood findings of some of the lab tests done on my guinea pigs.

I was totally outraged to learn that the upcoming RF studies (Senator Joseph Lieberman successfuly promoted release of funds for said studies), will not include testing of blood!!! I will be writing a separate letter in this regard and will send copies to you and Senators Lieberman and Wellstone. Dr. Ronald Melnick told me at the International Bioelectromagnetic Society's June (2001) meeting during a special session regarding RF's, that protocol for the RF testing had not yet been established BUT he went on to say that he did know that "no blood testing will be done!!" He offered the explanation that the tests are being set up for only 90 days and that would not be enough time.

My guinea pigs became sick and had adverse blood results after only two weeks of exposure (I would be glad to provide specific details to anyone requesting same). My two grandsons began to favorably respond to reducing their nighttime EMF exposures (due to sleeping next to "powerwall" -- they also live near high voltage powerlines albeit more than the 50 foot distance we are forced to live by), after two weeks of placing their beds on another wall.

Incidentally when I talked to Attorney Deborah Carney, who is a major spokesperson in regard to the Lookout Mountain case (telecommunications towers on the mountainside), she stated that there is a lot of dog leukemia out in Golden, Colorado. The people in the mountains surrounding Lookout Mountain (Golden) also have many health problems including various cancers. Her email was in response to my request for information regarding lymphocyte and neutrophil abnormalities (white blood cells) in that population as result of learning about the hematologic syndrome affecting my guinea pigs which represents a leukemic condition.

Asthma is another major concern in our family as well as the guinea pigs. I am well aware that our country is experiencing an explosion of asthma. Not every person who suffers from asthma will be sick due to EMF exposures; however, the evidence in this family clearly shows a very strong causal connection.

The list of health problems in our family is very long. The words "rare" and "unusual" appear often in our medical records. Strange blood changes are on lab reports in our records; however, our doctors do not even inform us because they don't understand what is going on. I learned even the veterinarians, who have been well aware of my EMF cocnerns, did not mention certain blood changes and it was only after I requested copies that I discovered this fact.

It is vital that something be done in the way of public service announcements in order to help keep others from developing chronic asthma, chronic sinus and ear infections, immune deficiencies and cancers. Chronic asthma, and sinus and ear infections were some of the symptoms my two grandsons suffered. The same symptoms affect many other family members. No genetic link was found in connection with the rare immune deficiencies and the subject of environmental problems was discussed to a limited degree until I brought up the subject of the powerlines.

It is my opinion that the American Medical Association should be apprised of the fact that blood changes are most likely due to environmental assaults and physicians should be required to advise patients of any and all abnormal tests no matter how insignificant or confusing the results may be.

The way to begin is for immediate action in regard to further EMF studies and a requirement that the public be informed of "possible" health problems from close proximity to EMF particularly in regard to electricity in close proximity to beds of children. Childrens' cells are in the developmental stage!!! How many more are we going to allow to endure the pain, agony and fear not to mention extreme financial burdens of those who develop immune deficiencies that subsequently allow leukemia and other cancers to develop, all because our government cares more about catering to the interests of "big money" than the lives of its' citizens???

Sincerely,

Joanne C. Mueller
Founder of Guinea Pigs R Us
731 - 123rd Avenue N.W.
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Email: JCMPelican@aol.com

cc: Senator Joseph I. Lieberman
Senate Hart Office Bldg.
Washington, DC 20510-2303

Senator Paul Wellstone
136 Hart Senate Office Bldg
Washington, DC 20510-2303

Enclosures: (1) Copy of Microwave News, Vol. XXI No. 1, "A Report on Non-Ionizing Radiation, Jan/Feb 2001, pps. 1-3 re "Federal Agencies Report to Congress: EMFs May Present a Leukemia Risk

(2) Guinea Pigs R Us information letter provided as part of a private exhibit at the International Bioelectromagnetic Society Conference, June 10, 2001

(3) Bovine Practitioner, Sept. 1994, "The Effects of Ground Currents on Dairy Cows: A Case Study" (note particularly highlighted portion on page 75 re lymphocytes and neutrophils and possible breakdown of immune system and/or possibly dangerous precancerous condition)

(4) 3-17-99 email to Roy Beavers (EMF Guru) titled "Letter to Clinton [President], et al

(5) 6-12-99 email to Roy Beavers (EMF Guru) titled "30 years of agony"

(6) State of Michigan, Attorney General's Office, letter to Blair, Wisconsin, electrical contractor, Dave Stetzer, dated 4-17-01

An Evaluation of the Possible Risks from Electric and Magnetic Fields from Power Lines, Internal Wiring, Electrical Occupations and Appliances
Draft 3 for Public Comment

Reviewer: Herbert L Needleman MD
Professor of Psychiatry and Pediatrics
University of Pittsburgh School of Medicine
September 4, 2001

I have reviewed the above document and submit the following comments:

This document is an unusual, if not unique approach to estimating a formidable problem: the hazard to health from the mixture of electromagnetic fields. I have been involved in a number of hazard evaluations in my position as a member of the EPA Science Advisory Panel for Pesticides, but have not encountered this approach in a regulatory context, or anything close to it.

The evaluation is innovative, cogent, evenhanded, comprehensive and scholarly. Earlier efforts to make sense of complex data sets in environmental health began with narrative reviews, and later progressed to meta-analyses. This evaluation is a large step forward. Recognizing that everyone brings a prior set of beliefs to the assessment of a problem, it explicates the beliefs of the three evaluators, roughly quantitates them, and employs Bayes theorem to update their priors. The evaluators were trained in probability estimation, another innovation, and then confronted with the data. This is a huge improvement over the conventional narrative reviews, in which the reader must infer the positions of the reviewers.

The transforming of private hunches into public opinions is unquestionably progress. It permits the priors and the impact of newer data on these judgments to be examined and in turn evaluated.

I am not familiar with this literature and cannot comment on the thoroughness of the review of studies. It is large and appears to be up to date. The studies and summaries are well organized and accessible. The explanation for not including clusters is persuasive.

Most impressive is the examination of the implications of "small" effect sizes—in the range of relative risks of 1.2 to 1.5, and the avoidance of Type II errors derived from making null conclusions from studies of little power, or failure to obtain complete consistency from groups of studies. The report has a sophisticated response to these solecisms.

The summaries take the form of a dialog, in which the arguments against causality are placed along the arguments for causality and the judgment of the three scientists stated. This is most edifying and helpful.

To respond directly to the questions raised in the cover letter:

1. Good data trumps theory every time. I agree that the null assertions by physicists have little authority.
2. I found the explanation for the priors persuasive. One does not often encounter an evolutionary explanation for an expected toxic response. It was very useful.
3. Lack of mechanistic understanding should not inhibit judgments about the reality of an effect, witness John Snow. We do not know the mechanism for lead's impact on the brain, but we know it happens.
4. Animal studies can support a theory, but cannot invalidate it, particularly if the number of subjects is 100 or 200.
5. I thought your explanation of the importance of small RR was correct. The consistency of positive reports is quite strong, and the population exposed is quite large.
6. I agree.
7. I thought the presentation of the arguments was strong. (see above) I have not encountered this format before.
8. I have no difficulty with this classification.

CONCLUSION: This is an extremely valuable and sound analysis of an extremely contentious and difficult problem. I would like to see a glossary of terms in the front pages. I can find no other fault with it. To the contrary, I learned a lot and was persuaded by what I learned. It is not overblown to say that this report, using a Bayesian framework, enables the reader to come much closer to the decision making process, and makes public forces that have otherwise been submerged. This review marks a new departure in data synthesis, and may lead to many such efforts in other areas. The authors are to be congratulated.

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December 7, 2001

Dr. Diana M. Bonta
Director
Department of Health Services
1515 Clay Street, 17th Floor
Oakland, CA 94612
USA

Dear Dr. Bonta,

I am writing this letter to you because I have had no response to the questions that I asked on the comments of your home page on November 29 and December 2, 2001, regarding an approximate date when your final report on the health effects of exposure to power frequency electric and magnetic fields will be made public.

I have read your draft 3 for public comment, April, 2001, "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances" with great interest. I am convinced that this report goes far further than anything available anywhere else in the world.

Here about 100 meters distant from my house Kansai Electric Company is now in the process of constructing a power line. In this area the frequency is 60 Hz and the voltage of the line is 154 kilo volt. I have been asking the company to take measures to prevent the health hazards from occurring. I have suggested that they should consult your report. Kansai Electric Company, however, disregards your statement for the reason that the draft is not final. I would like to ask you, in this connection, approximately when you are going to make your final report public.

Sincerely yours,

Kazunori Oshima
Former Professor
Doshisha University
Kyoto, Japan

Review of Electric and Magnetic Field Risk Evalation and Policy Options document of July 9, 2001

Reviewer: David Ozonoff, MD, MPH, Professor and Chair, Department of Environmental Health, Boston University School of Public Health

Overall comments: This review of the scientific evidence on the health effects of low frequency EMF is the Cadillac of reviews, not just of EMF but of scientific reviews in general. It exceeds in thoroughness, thoughtfulness and sophistication any other review by a wide margin. Indeed, there are no comparable efforts which I am aware of on any subject.

The use of the Bayesian framework is especially appropriate for reviews of this nature. Most scientists, consciously or unconsciously, use Bayesian approaches to evaluating evidence, and the systematic recognition of this in the review under consideration enables the most coherence and compatibility with how scientific judgments are actually made. Moreover the care with which this was undertaken adds immeasurably to the product, making it not only a demonstration of principle but especially pertinent and useful.

I strongly endorse the scientific methods and reasoning used here, independently of any conclusions. It is outstanding.

Answers to specific questions:

9) Underlying physical and biological models.

I agree completely with the approach that claims of "impossibility" or "implausibility" based on underlying physical or biological models should not be determinative or even greatly influence the evaluation of the evidence. I think many of the specific reasons given in the text are completely valid and I endorse them. I do not agree with the more general reason given in your specific question that theories should be used to predict results that are falsifiable. This idea (that science is "demarcated" by its production of falsifiable statements) has long been abandoned by philosophers of science (it belongs to the last time there was anything even approaching consensus in the field which was more than 40 years ago) and the text of the Review makes clear why it doesn't work: "falsifiable" predictions made by the theory do not really falsify anything. The existence of EMF bioeffects would not falsify electromagnetic theory, only suggest that some assumption or background condition is incorrect. We need to get by the "falsification" canard in epidemiology.

2. Animal bioassay, introduction of electricity

I agree (for the reasons given in the text) that animal bioassays should not be determinative here. The effect of the introduction of electricity on various rates, besides being subject to all the qualifications given in the text, represent a type of ecological design known to be subject to severe bias from confounding and effect modification, factors which are especially pertinent over the long time spans considered here. Thus such arguments are essentially useless (you can refer to the work of Morgenstern, Greenland, Robbins, etc., etc., here).

3. Influence of mechanistic explanations

Again, I agree completely and strongly with the approach taken here. The influence of knowledge of mechanisms is (as said) asymmetrical. Indeed it is my view that if and when such a convincing mechanism is produced there will be essentially an end to any EMF "controversy." On the other hand, if such a mechanism is not produced, the controversy will continue, unabated. This is validation of the approach taken here.

4. Pathology

This is connected with the argument about mechanisms and is subject to the same reasoning. I strongly agree with the approach here.

5. Relative Risks below 2.0

With a highly prevalent exposure such as EMF it makes no sense (scientific or public health) to ignore "weak" effects. Indeed it is not agreed as to what constitutes a "weak" effect, since many investigators (e.g., Monson) consider effects above 1.5 moderate. I agree with this characterization, and moreover, as many texts emphasize, many causal effects are not large. In evaluating such effects one must (as was done) consider the likelihood that bias or chance produced the result. But this is taken into account with your method.

6. Specificity

"Specificity" as an attribute of causal associations has long been disregarded by epidemiologists (see, for example Hill's own paper and many texts, e.g., the new edition of Rothman and Greenland which considers it essentially "useless.")

7. Transparency

I think the presentation is outstanding in most respects. It is less clear only in the area which describes how the actual final degree of confidence was arrived at. I think some more words might be useful here. Description of the "pros" and "cons" is unusually thorough, thoughtful and sophisticated.

8. Terminology

I think the division between "Virtually certain" and "Highly probable" is problematic. Isn't what you mean "I'm quite confident it's causal" and "I'm pretty certain it's causal" and not much more? Trying to assign numerical values here is pretty meaningless. Both of these categories are in the "above 90%" category, and trying to find a border in that range doesn't really mean much.

Minor comments on the text:

Page 25, section 2.2

I think you might note the issue of bias in ecologic design here (lines 5ff). Ask Morgenstern. In addition, Greenland has written several articles on the problems of attributing PAR in the face of ignorance of causal mechanisms. I think this is especially important here in terms of the uncertainty if EMF is an initiator or promoter. I am troubled by the PAR calculations as they have additional assumptions noted in Greenland's various analyses.

Page 28, lines 2 – 5

The fact that "causality" is not empirical (but a judgment) or analytic (i.e., subject to "proof") is vitally important and should be emphasized. I agree completely.

Page 28, lines 39 –43

We are also dealing with complex, non-linear systems very unlike the linear physical systems the physicists views are based upon. It is known that such systems often behave in ways very different than might be expected given the usual analyses (e.g., sensitive dependence on initial conditions, possibility of chaotic behavior, hysteresis effects, etc.).

Page 64, lines 31 – 34, and also found elsewhere in the Report, e.g., page 68, lines 31 - 35

It is not obvious to me why avian data are not applicable. Maybe there is better data, but one of the main reasons chick embryo studies are done is to shed light on mammalian teratogenesis and reproduction. They are obviously somewhat informative in that respect. This rejection seems much too strong to me.

Page 65, table 7.1 and in the text, various places

These "attributes" (Hill called them "viewpoints") are different than the ones used by Hill, as is the terminology. Moreover, I think it has been shown (see Weed's papers) that most of them are not used by epidemiologists in the course of usual practice. They are appropriately used in this Review, but the implication that this is commonly done is wrong. I don't think you need to attribute them to Hill, but if you do you should signal that your list is different in content and emphasis than his.

Page 67, lines 44 – 51

The use of statistical tests not only assumes "no association" but also "no bias." This is extremely important and should be added.

Page 68, lines 36 – 41

The statements made here are completely true (goes to a point originally made by Quine and is diametrically opposed to Popper) and shows that the original statement in the Report (commented upon above) is incorrect. If the negative studies don't "falsify" the original hypothesis (and they don't), than falsifiability is meaningless as a general proposition.

Page 69, lines 9 – 18 (Temporality)

As noted by Rothman and Greenland in their discussion of the Hill viewpoints, lack of temporal sequence only rejects causality in that study. It is not a rejection of causality in general, say in other studies where the sequence is appropriate.

I hope these comments are useful. Let me reiterate that I think this is an outstanding effort, far better than any other review I have ever seen, and an extremely important contribution to the scientific dialog.

David Ozonoff, MD, MPH
Professor of Environmental Health
Chair, Department of Environmental Health
Boston University

Comments on California EMF Program Risk Evaluation, Draft 3
Charles P. Quesenberry, Jr.

Dear Dr. Neutra,

Below are my responses to the questions posed in the letter of 7/9/01, and in many instances are similar to my previous responses in my review of Draft 2. My page and line-specific comments and suggestions on previous drafts have all been addressed, and I have noted no further issues in Draft 3. Thank you for the opportunity to contribute to these efforts.

1. The position taken by the reviewers of not being greatly influenced by arguments for zero probability of EMF effects at residential and occupational levels seems reasonable, and adequately justified in this document.
2. I felt that the reviewers' final choices of the prior distribution are similar to what I would have chosen. I cannot recommend any changes. The development material in Chapter 2 is well presented, and useful in interpreting their decisions. The three sets of arguments leading to their choices are reasonable.
3. My review of Chapter 4.7 of the NIEHS Working Group Report leads me to agree with the position that convincing mechanistic explanations do not currently exist. However, your arguments as presented in Chapters 4 and 5, and elsewhere, supporting your position that this does not greatly influence your level of confidence in EMF health effects are generally well reasoned and convincing to this panel member.
4. Similarly, upon review of the NIEHS report, your summaries in presented in Chapter 6, and appendix, your position with respect to influence of the animal pathology findings on your level of confidence is reasonable.
5. I do not believe that the position taken with respect to evidence for confounding or bias in interpreting relative risks less than 2.0 is overly problematic, particularly in the context of this evaluation of the various aspects of the stream of evidence from multiple studies for each endpoint under consideration - number of studies, consistency, dose-response, heterogeneity in populations studies, occupational vs residence.
6. Your position with respect to lack of specificity in association is reasonable, and generally well argued in chapter seven. The argument for evidence regarding the association with one endpoint influencing the confidence in association with another endpoint is reasonable, primarily in the weakening of the basic argument of environmental exposures to EMF are at too low of a dose to result in health effects - as presented in chapter seven.
7. Generally, the quality of the pro and con arguments seems balanced. Occasionally, an argument seems somewhat weak, but this was not the overall impression of the presentation.
8. The only way I can think of eliminating the overlap issue in the current phrasing of confidence is to either a) remove the ">50%" and "<51%" from the phrases, and reword to something like "Moderately probable", and "Moderately improbable"; or b) always include the range of certainty. I agree that the overlap with "Possible <51%" and "Possible > 50% is problematic. I suggest either using <=50% or < 50%.

Mr. Jack Collins
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Division of Environmental and Occupational Diseases Control
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Basel, 30. 8 2001

Concerns: Third Draft of EMF Risk Evaluation and Policy Options

Dear Mr. Collins,

Having received the draft only today, I restrict my comments on some points I feel able to answer without consulting the large body of literature on EMF or positions papers existing. I thank you for the possibility to do this and hope my response will be in time and useful.

Yours sincerely

Regula Rapp

Questions to the reviewers:

Point 3: I am not convinced that knowing mechanistic explanations for EMF-effects are absolutely necessary before making policy decisions, or even before judging about causality. There are several examples of excellent epidemiologic evidence for causality before evidence from experimental studies existed (e.g. the famous Cholera epidemic in London, where Dr. John Snow removed the pump handle in Broad street and stopped the epidemic, suspecting dirty water as cause without knowing even the existence of bacterias).

Point 5: I agree that relative risks between 1 and 2 should be taken seriously. Today, almost everybody is everywhere exposed to low frequency EMF, so you can't expect a wide variation in exposure. If you compare the risk of smoking 20 cig/d with the risk of smoking 25 cig/d, you will not get a big risk estimator, nevertheless the overall risk of smoking is high.

Remarks to the draft 8.0 epidemiology of the leukemias, adult leukemias:

The biggest problem of the mentioned studies involves exposure measurement. The Swiss study on railway engineers, published recently also in English, has in my opinion the most reliable exposure measurement ever performed due to the long use of the same electric driven engines, (lasting for decades) where fields can be measured nowadays. The authors found a dose-response relationship for magnetic fields to leukaemia mortality of 0.9% (95%CI 0.2-1.7) increase in leukaemia mortality per μT-year of cumulative exposure or of 62% (15-129%) increase per years spent under exposure of 10μT and higher.

Minder CE, Pfluger DH
Leukemia, brain tumors, and exposure to extremely low frequency electromagnetic fields in Swiss railway employees
Am J Epidemiol 2001; 153 (9): 825-35

Dr. DA Savitz' questioning the results that they do not apply to risks of the general population due to the lower frequency field of 16 2/3 Hz was answered by the authors, that resonance effects (as calcium ion concentration alterations in cells) observed at 16 2/3 Hz mean that resonance will occur with exposures at or near low multiples (60 Hz) of this frequency as well.

Savitz DA
Invited commentary: electromagnetic fields and cancer in railway workers
Am J Epidemiol 2001; 153 (9): 836-8

Minder CE, Pfluger DH
Minder and Pfluger respond to "Electromagnetic fields and cancer in railway workers" by Savitz
Am J Epidemiol 2001; 153 (9): 839-840

In my personal view the risk of magnetic fields for leukaemia in adults are 90 to 98% likely causal. As the exposure in the Minder-study was really very heavy, the additional lifetime risk in the population will be very very slight, perhaps we will never be able to demonstrate it in an epidemiological study.

It should not be anticipated, that children are more susceptible to the risk of leukaemia than adults. In the case of benzene, there is even some evidence for stronger effects in older people.

Risk Evaluation and Policy Options
By The California Department of Health Services
Electric and Magnetic Fields Program

Comments by Cindy Sage
Sage Associates
1225 Coast Village Road, Suite G
Santa Barbara, CA 93108

DHS should be commended for its tenacity and even-handedness in conducting the California EMF Program and in dealing with multiple stakeholder interests. As a member of the original Stakeholders Advisory Group for the California Public Utilities Commission EMF Consensus Group (1991-1992) I am pleased to see the report has been completed.

Twelve years have elapsed since the California DHS began its first childhood cancer cluster studies where EMF was explored as a potential risk factor. Children in those elementary schools in 1989 are now old enough themselves to be in medical school. The California PUC EMF Consensus Group report originally urged "no and low-cost" measures to reduce EMF exposure in early 1992. In late 1992, the Swedish and Danish studies on transmission line exposure and childhood cancers confirmed 2 to 3-fold risks above 2 mG. Many studies have since reconfirmed the findings of these early epidemiology studies. The 1998 NIEHS Working Group Report recommending that EMF be classified as a Category 2B carcinogen, and the recent unanimous WHO IARC vote to classify EMF as a Category 2 B carcinogen have all reinforced the need for California and the US to move forward with regulation and policy on this public health issue. California's policy in need of serious updating in light of the new DHS EMF Program Report.

The harm from delay in providing "prudent avoidance guidance" to the public during these years cannot be undone, and what adverse consequences that may have resulted are beyond rectification now. Some stakeholders in 1992 considered the "weight of the evidence" sufficient for DHS to provide public advisories on EMF exposure. This was not done then. It should be done now, and regulation of EMF as a carcinogen (whether Category 2 B or higher classification at 1B or 1A) should be pursued on an urgent basis.

The following observations and recommendations are offered as comment on the Draft DHS EMF Report:

- That DHS recommend to the appropriate state and federal agencies (EPA) that regulatory action on EMF exposures be adopted, and that DHS provide recommendations for public health exposure standards in line with other carcinogens that are regulated.

- That DHS notify the State Department of Education School Siting Board of its findings and assist them in revising their EMF setback policies for schools. Both new school building and existing schools that are building new classrooms under power lines should be included in revised standards for setbacks. At present, existing schools are exempted from the EMF setback guidelines.

- That DHS establish a rationale and recommendations for a public exposure limit for EMF at 1/50th of the magnetic field level linked to increased risk of childhood leukemia (in keeping with ANSI/IEEE standard-setting protocols).

- That DHS re-evaluate its attributable risk for leukemia in light of the recent paper published in Medical Hypotheses by Sam Milham (2001).

- DHS should be commended for its efforts to consider the latest relevant epidemiological literature. It would have been unwise to disregard pertinent literature by using artificial cut-off dates that would exclude important study findings.

In this light, DHA should continue to monitor studies on breast cancer that are due to be published shortly and review both conclusions and the underlying data in order to update its level-of-confidence evaluation on male and female breast cancer. Nine of ten published epidemiological studies on male and female breast cancer report positive linkage between EMF exposure and risk. Further, there is supportive evidence from both animal studies (Loscher, Mevissen) and from cell studies (Harland & Liburdy, Luben, Blackman).

• DHS should be funded and authorized to update its level-of-confidence evaluations on EMF exposure and health risks, and report back on an annual basis with written suppplements to the DHS EMF Program Report.

• An important premise used in the policy analysis that must be clearly articulated to give context for its conclusions is the choice to consider costs associated with fixing existing infrastructure to reduce EMF, and not the much lower costs associated with prevention of new EMF exposures from new construction. Otherwise, a very unfavorable cost comparison is generated. It takes more deaths to justify fixing the existing infrastructure (you have to knock down the old, remediate or rebuild). The cost to build a new transmission line with lower EMF exposures (bigger setbacks to sensitive receptors, undergrounding, field cancellation in overhead conductors, etc) is far less expensive than retrofitting.

• DHS should request the CPUC to notify all investor-owned utilities (and give advisories to municipally-owned utilities) to avoid building new electric facilities that would generate EMF exposures along ROWs in excess of magnetic field levels associated with increased risk of any cancer or other adverse health outcome identified by DHS in this Report. Further, once DHS has specified a regulatory limit (1/50th of the level of magnetic field linked to increased health risk is recommended), it should so advise the utilities to plan new facility location and design to incorporate such EMF limits.

• Utilities should be required to notify in writing any homeowner, school, day-care or pre-school, convalescent home, hospital, clinic, mental health facility, recreational facility, park, and other habitable building of levels exceeding 2 mG of the actual EMF level due to power lines at that site, and provide a copy of the DHS EMF Program Report Executive Summary.

• DHS should notify every local jurisdiction's planning department to consider an update of its general plan and zoning regulations to take EMF into account in establishing setbacks for habitable buildings, parks and recreation areas.

• The Policy Options section (Table 2) shows that for schools, the cost to fix net current problems is $16 of the total of $43 million total (which also includes the cost to fix electric panels, distribution and transmission lines). The cost to eliminate net current problems should not be charged off against EMF mitigation costs. It should be excluded in this discussion. These problems are almost universally caused by National Electric Code (building code) violations. These violations create spark, shock and fire hazards and should be corrected anyway.

• The Policy Options section states "there could well be anxiety generated by mandated avoidance action in the school, power grid or home grounding sectors." I agree that anxiety itself may be harmful to health, and this long period of inaction, disinformation and uncertainty has indeed been harmful to those whose lives, investments and property values have been "in limbo", or who have sustained injury without possibility of legal relief.

However, I think DHSs concern here is misplaced. DHS should well hope that the public even pays attention. For over a decade, the public has been sedated with false assurances of safety, by "science-by-press-release", by selective and biased reporting and exceptionally poor media coverage of EMF issues. Now, it is more likely the public will react to this decade of ambiguity with issue exhaustion, confusion, disinterest, and distrust of governmental authority. However, if DHS can finalize this report, notify affected parties within the State, and act in a continuing capacity to oversee and assist in regulatory action; it should be possible to take prudent actions to lower existing EMF exposures and avoid new ones.

September 10, 2001

Dr. Raymond Neutra
California EMF Program
1515 Clay Street, Suite 1701
Oakland, California 94612

Dear Dr. Neutra:

I am writing to provide public comments on the California Department of Health Services (CDHS) *draft* report entitled, "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances." The views expressed in this letter are solely my own and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas and Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District and San Diego Gas and Electric Company.

My comments are attached. I have valued the opportunity to participate in the California EMF Research and Education Program. Please contact me if you need additional elaboration. My CV is attached. I appreciate the opportunity to provide these comments.

Sincerely,

Jack Sahl

Written Public Comment on CDHS *draft* EMF Report:

An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances

Submitted to:

California Department of Health Services
California EMF Program

1515 Clay Street, Suite 1701
Oakland, California 94612

Jack Sahl, Ph.D.
J Sahl & Associates
330 North Indian Hill Boulevard
Claremont, California 91711

The views expressed in this letter are solely my own and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas and Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District and San Diego Gas and Electric Company.

September 10, 2001

My comments are in three sections[1]. In the first section, I respond to specific text in the Executive Summary and Chapters 1 – 7. In th
+e second section, I provide comments on Chapters 8 – 19. In the third section, I respond to the specific questions posed by CDHS (1 – 7).

Comments on Executive Summary and Chapters 1 – 7

Executive Summary

Section 2 – A Summary of What Has Changed <Pages 5 – 7>

Page 5, Lines 48 – 59, Biophysics

You do not correctly apply the available biophysics information. You first make the argument that the relevant biophysical theory (i.e., based on information gained from observation) shows that EMF effects are impossible. Since you reject that EMF effects are impossible, you then inappropriately discount the available biophysics information that would prove useful to the evaluation of the experimental laboratory, whole animal bioassay, and human epidemiology. The proper question to ask is not whether the available biophysics information proves that EMF exposures do not cause disease, but what is the strength of evidence that they do. For this question, the information from biophysics is highly relevant to the evaluation of the available data.

Page 5, Lines 60 – 67, Mechanistic Research

There are established biophysical mechanisms for how ELF-EMF can introduce either magnetic fields or electric current into human cells. The observations on which these mechanisms are built are reproducible in laboratories around the world. It is likely that new information about how EMF alters chemistry within cells or tissues will help to refine and extend these established mechanisms. It is very much less likely that an entirely new mechanism will be discovered. There have been extensive efforts to find a mechanism to explain biological effects at field strengths commonly found in the residential and occupational environment (from between 1 – 100 mG). These efforts have failed, not from a lack of trying, but because the data do not support them.

Page 6, Lines 24 – 29, Epidemiology

You overstate the significance in the apparent growth in the epidemiology literature with respect to sudden cardiac death, dementia, suicide, and spontaneous abortion. I will respond in more detail in the specific chapters, but it is important to acknowledge that this literature is very weak at this time.

Page 6, Lines 38 – 66, Aspects of the EMF Mixture

You overstate the complexities of the physical aspects of EMF as it pertains to an evaluation of the existing literature. While I agree that this might be interesting if you wanted to design and fund future research, it is not particularly relevant to an assessment of risk based on the currently available scientific literature. Your risk assessment should be conducted for specific exposure-disease models. For example:

[1] I would like to acknowledge the valuable contributions of Dr. Ken Foster in helping to clarify the biophysical science associated with power-frequency electric and magnetic fields.

Question 1: What is the evidence that 50- and 60-Hertz magnetic fields cause childhood leukemia (and then, individually, any other disease)?

Question 2: What is the evidence that 50- and 60-Hertz electric fields cause childhood leukemia (and then, individually, any other disease)?

Questions 3 – 99: What is the evidence that any other aspect of ELF EMF exposures cause disease (specified by type)?

There is very little information to address these proposed questions 3 - 99, and a formal risk assessment would not prove productive.

My guess is, however, that a fair reading of the available information about 50- and 60-Hertz exposures, given the established biophysical models, would prove reassuring with respect to potential health risks. This is due to our current understanding that the amount of energy in this alternative exposure conditions is much lower than for 50- and 60-Hertz electric and magnetic fields (accept possibly for 'contact currents' and 'transients'). Please note that no amount of speculation about novel biophysical mechanisms for these exposures should strengthen or weaken the assessment of exposures to 50- and 60-Hertz electric and magnetic fields.

Section 4 – The Weight Accorded To Streams of Evidence By The Reviewers <Pages 7 – 8>

Lines 18 – 21, 'Biophysics'

I agree that the 'established physical theory and simplified biological models' are not sufficiently strong to **prove** the impossibility of the epidemiology. This information, is however, clearly relevant to the evaluation of the epidemiological literature and assessing the predictive value of whole animal bioassays for human health. Your presentation of this argument is tantamount to saying that since the epidemiology literature **does not prove** that magnetic field exposure causes cancer, the available data are not useful in the evaluation. I reject both sides of that coin, and urge you to include all of the available information to evaluate your specific exposure-disease model. Where is the biophysics expertise? If Jim Weaver were an author, do you think this report would be worded like it is (Dr. Weaver is a respected biophysist at MIT/Harvard who has published many papers in this area)?

Lines 41 – 46, 'Experimental Evidence'

You ignore the vast laboratory experimental literature that did not find any biological effects. Much of this first started from initial reports of robust effects, which could not be replicated in the original lab or in other labs. The key parts of this literature are directly related to carcinogenesis. The fact that no supporting evidence emerged after the focused research funded by NIEHS EMF-RAPID is clearly relevant to the evaluation of the epidemiological data.

Lines 47 – 61, Page 7; Lines 1 – 2, Page 8, 'Animal Pathology Evidence'

The authors take the position, that while the whole animal bioassays have shown no carcinogenic effects, the relevance of this information is diminished since:

a) 'there are known human carcinogens for which no animal existed for decades'

b) ‘whether the bioassay of one element of a mixture could be sensitive enough to detect problems for the entire mixture;
c) ‘questioned the sensitivity of a bioassay involving a small number of animals and assuming a monotonically increasing risk from low to high dose, when the epidemiological studies that prompted the bioassays did not suggest this was so’.

Furthermore, they found supporting evidence due to:

d) ‘repeated, but unreplicated results … that showed co-promotional effects on breast tumors’; and
e) ”non-robust effects of pulsed EMFs on chicken embryos in a number of laboratories”

As to (a), the difficulty with finding an appropriate animal model for agents like ‘cigarette smoking’ was that it was difficult to get the dose to the relevant site. For ELF-EMF, the external exposure naturally leads to a dose to the whole body, thus this objection is overcome. Another problem is with the ingestion and metabolism of the agent of interest. Again, this is not a consideration here given the physical nature of the exposure and that our question of interest is not metabolism, but rather coupling, signal transduction and bioamplification of the signal. There is no suggestion that this would be different for a rat, mice, or human cell/tissue. As long as you do not ignore the biophysical evidence for how fields couple and impact chemistry within cells, the best evidence is that rats and mice should receive a dose from external fields just as humans do. Also note the important consideration about ‘scaling’ based on the size and posture of the test animal. This reduces the resultant dose for rats and mice, but again, makes it more like our human environment (plus studies which turn the field on/off and changes in field orientation as the animals move around the cage).

With respect to (b), this cannot be addressed unless your risk assessments are based on a specific exposure-disease model. If the agent is induced current from 50- and 60-Hertz electric and magnetic fields, then most of the ‘mixture’ in the environment that you find confusing, only influences the coupling to the cell and therefore, the dose of induced current. If all these ‘ingredients’ in the human environment act to subtract from our exposures, then we should be comforted. If they add, then our studies of animals are all the more relevant because of the high external exposures. For the rats and mice that were not electrocuted in the earlier studies, or warmed up, the fields they experienced were very much higher than normally found in our homes or workplaces. But the mixture was similar in quality in that the fields are not uniform, the change in orientation due to the movement of the animal in the cage, they are turned off for cleaning and feeding, turned on both night and day. (The above discussion is also relevant to other established biophysical models, including magnetic fields acting directly on a cellular receptor). If your exposure-disease model is different, then you should evaluate this separately. But the lack of data for your non-specified model cannot detract from the value of the extensive whole animal bioassay data that consistently show no biologic effect or evidence for cancer.

For (c), the experience has been that whole animal bioassay’s perform well by using the Maximum Applied Dose (MTD) to balance against the small number of animals. But without evidence for a particular dose-response model, why should these tests have been funded? There is no evidence for a plateau in the adult leukemia studies for men occupationally exposed. The childhood leukemia data suggest a ‘threshold’ at 3 – 4 mG, but after that it is all too sparse to tell much of anything. Note that the available literature does not use the MTD for magnetic fields. Rather, a range of exposures are applied that are designed to be high to very high relative to what is found in the human environment.

With regards to (d), neither NIEHS, NRPB, nor IARC found the German or Soviet studies compelling or relevant to cancer promotion.

With regards to (e), the 'Hen House' data are not consistent in their results, are not considered compelling, do not represent exposure conditions found in our environment, and are not relevant to the diseases evaluated in this draft.

Section 5 – Degree of Confidence of Causality and IARC Classification After Considering All Streams of Evidence <Pages 8 – 12>

Table 1. 'Degree of Confidence Table'

A fair reading of the available scientific information does not support the results presented in this table. Your IARC category does not result from the same procedures as used by IARC. The 'Confidence in Causality' are not based on a defined 'exposure-disease model' and therefore have no scientific basis. Given the currently available information, it is implausible that the different categories are meaningful. From the text in the report, it appears that the reviewers included both the currently available information and their expectation of how future research will turn out. The 'Degree of Confidence…' is not representative of the conclusions from other independent assessments (based on the same literature but using standard methods to assess the literature and a more representative panel of experts),

Section 6 – Disparities <Pages 13 – 15>

The value of having a report on potential EMF risks published by the California Department of Health Services (CDHS) is that you want to represent the current scientific literature. The views of the three authors of the draft are not in line with those expressed by international panels of independent experts. This report does not provide the necessary justification to diverge from the positions of the U.S. National Institutes of Environmental Health Sciences (NIEHS), United Kingdom's (UK) National Radiological Protection Board (NRPB), and the World Health Organization (WHO) International Agency for Research on Cancer (IARC). The same scientific literature was available to all four groups (except for the recently completed studies on miscarriages). Each of these four panels concluded that the strongest information is for childhood leukemia and exposure to power-frequency electric and magnetic fields (EMF). However, there is insufficient scientific explanation for the huge differences in their assessments.

Section 7 – How The Degrees of Confidence And Ranges of Uncertainty Could Be Used In Policy Analysis <Page 15>

There should be a clear separation between the assessment of potential risk and developing inputs for 'policy analysis.

Section 8 – What Aspects Of The EMF Mixture And What Summary Exposure Metric Might Be Bioactive? <Pages 15 – 16>

Please refer to the relevant discussions in response to other sections of the draft report.

Section 9 – Considering The Range Of Exposure In The General Population How Much Morbidity and Mortality Might Be Attributed To EMF's <Pages 16 – 17>

Since your underlying assessment of risk does not specify an exposure-disease model, and the report consistently over-estimates the estimated risk, it is not likely that these presentations are helpful.

Section 10 – If EMFs Were Hazardous, What Would Be The Probability Of Developing Disease in Persons Who Were Highly Exposed? <Pages 17 – 18>

Since your underlying assessment of risk does not specify an exposure-disease model, and the report consistently over-estimates the estimated risk, it is not likely that these presentations are helpful.

Section 11 – Policy Relevant Areas For Further Research <Pages 18 – 10>

The report states that: "…if one were serious about clarifying this issue, there would need to be a long-term commitment to steady research funding and funding for intermittent assessments of the state of the science…"

The electricity supply industry and the U.S. government have been funding EMF health research for over 30 years. There have been more than 75 reviews of the scientific literature by expert panels over this period. The national EMF-RAPID program was funded specifically to confirm any biological effect. Most of this research is focused on 50- and 60-Hertz electric and magnetic fields. This is because these fields are the dominant source of exposures for ELF-EMF.

The hallmark of this research effort has been to have preliminary results suggesting a problem. Upon follow up with better studies, the first set of data has not been confirmed. This is the reason why the senior investigators at our nation's best universities and research centers are not submitting applications for funding from the established NIH peer-review grant process.

Chapter 1. Introduction

Section 1.3 Supplemental Degree of Confidence Approach To Evaluation

Pages 21 – 23

Each of the Reviewers gives too much weight to their belief about the results from future research. Each believes that since EMF is 'more likely than not to be a health hazard'; they naturally assume that future research will only increase the weight of evidence. This 'bias' is inescapable when you do not limit your assessment to the existing literature. Throughout the draft, the Reviewers consistently assume that when there is a lack (or limited) information, that once the necessary information is obtained (e.g., with future research), that this will support the belief in causation. This is tantamount to turning scientific ignorance into knowledge supporting EMF risks. This is especially problematic in this area, since the pattern of thirty years of intense research is for the results from first generation studies to be not supported by the results of more rigorous follow-up research. With regards to a science-based 'risk assessment', you should be neutral about the expectations of result from future research. In policy debates, you could argue that it would be appropriate to be cautious in the face of this uncertainty. It is essential to keep these two considerations separate to achieve the best public health policies.

Chapter 2. The Initial or "Prior" Degree of Confidence of a Possible EMF Hazard

Section 2.4 – Conclusion of the Core Evaluators <Pages 29 – 30>

Researcher 1, Lines 21 – 42

Researcher 1 states that: “In this case, we are justified in believing that an increase from virtually zero to several mG represents a massive increase in dose that is not easily tolerated.” This is based on the conception that EMF represents an ‘extraneous agent’ and that: “The probability that extraneous electrical signals leave an organism that depends on electrical signals for proper functioning totally unperturbed also is very small.” Furthermore, Researcher 1 “…has no basis to believe that repair mechanisms against an unknown and totally alien agent may have evolved by accident…” This line of reasoning fails because, while it could be argued that a 60-Hertz field is novel, the currents that are introduced into the body are not. It is the currents that are the relevant biological parameter (see below for thoughts on viewing the magnetic field as the ‘dose’ of interest). The frequency of the external field, along with other exposure characteristics, are relevant to the field’s ability to couple with the cell, to introduce current in the extracellular environment. Our cells and tissues evolved and thrive in a very rich and intense electrical environment. First, the exposure is measured in mG-units for the magnetic field, but to talk about ‘dose’ we need to rely on a specified exposure-disease model. In this construct, the current introduced from the field is the dose. Here, the current from the external field is a very small increment to that which already exists with our biological systems. Furthermore, our cells and tissues have evolved insulation properties to help manage electrical currents. This is all well known, based on experimental observation.

We know that our cells thrive in a sea of electrical activity. The next steps are signal transduction and signal amplification. To change cellular chemistry, these currents need to use established cellular machinery, with attendant feedback mechanisms. For Reviewer 1 to have a compelling argument for induced currents to have an effect, he also needs to explain how the induced currents distinguish themselves from all the competing signals trying to communicate with our cells. Thus, from a cell’s perspective, the signal from an external magnetic field is very faint, and has to compete with very much larger, normally occurring electrical signals to gain the attention of our cellular machinery. But the interesting question is how could a very small change of external field (i.e., from 3 mG to 4 mG for childhood leukemia, or ‘at least 8 mG once while pregnant) be sufficient to trigger a 10-80% increase in disease?

All of these arguments are supported by a vast literature based on observation and theory. They don’t prove that EMF has no impact on health. That is not the point. This information should be used to inform our evaluation of the experimental laboratory, whole animal bioassay, and human epidemiology.

Researchers 1, 2, and 3 can easily construct a hypothesis for an effect from other than induced current or magnetic fields for which I do not have sufficient data to refute. While may be a valuable scientific exercise, the mere existing of such hypothesis neither strengthens nor weakens our beliefs about causation for other exposure-disease models.

If you view the ‘magnetic field’ as the relevant dose, then you have to argue that the body has a receptor, or a ‘detector’ for magnetic fields to complete your exposure-disease model. It has been established that there are magnetic field receptors in other species (e.g., bees and birds use magnetite to assist with navigation, and do just fine around high voltage transmission lines). People have looked, but not found a plausible receptor in humans, rats or mice.

If it is not the induced electrical current or the resultant magnetic field, then what is the 'dose' that you propose to couple with cells to change chemistry to change biology to cause disease? If you don't know, how can you perform a risk assessment?

Chapter 3. The EMF Mixture

The discussion about 'The EMF Mixture' is only relevant if you have specified an exposure-disease model. Otherwise it does not have value in the assessment of risk. It does inform our debate about future research and the value of 'cautionary approaches' for setting policy. Remember, the lack of information about a novel biophysical model should not be used to increase your confidence about the probability for a health risk.

Chapter 4. Biophysical Issues

You consistently discount the predictive value of the available biophysical data since 'they are theories not based on observation' (while I disagree with the characterization, since the 'theories' were based on data that has been largely accepted by the scientific community), however, note the terms used in the 'For Causality' column (Table 4.1.1):

(F1) "One cannot **anticipate** all the possible …
(F2) "…fields **may** exhibit spatial and temporal coherence that **may**…"
(F3) "…models have been proposed that **may** well be viable…."
(F3a) "Better personal exposure monitoring **may** show an effect."
(F4) "A physical agent **may** interact in more ways than one."

You use ignorance (which others with more knowledge might even disagree with) about biology to argue 'For Causality.' This is not right.

Chapter 5. *In Vitro* Mechanistic Studies

In Section 5.2, 'Pro and Con Arguments', Table 5.2.1, you state that the genotoxicity/mutagenesis data argue against a cancer initiation model. Why don't you conclude that we have ruled out cancer initiation as the relevant exposure-disease model?

Chapter 6. Animal Pathology and Physiology

My points, made in other sections of the report, can be summarized by:

1. Results from RTP whole animal bioassays are relevant to humans
    a. Exposures provide dose to site of interest
    b. Biophysical coupling will be similar (gross electrical properties of rat/mice body, tissues, and cells will be the same)
    c. Signal transduction will be the same. But maybe not…if not, then mechanism for bioamplification/biofeedback would act to mitigate signal amplification
2. The dose in the whole animal is representative of the dose in residential and community environments
    a. High doses cover the mixture if any established biophysical mechanism is relevant
    b. Scaling lowers the effective dose if you argue for a step dose-response function
3. The whole animal bioassay have used the relevant biological model:
    a. If initiation, the mutagenesis data and the whole-animal bioassay show no general cancer or leukemia effect

   b. If promotion, the radiation/DMBA initiation MF promoter leukemia and lymphoma studies are relevant
4. The CDHS review did not include the relevant data from Japanese studies
   a. General cancer
   b. Leukemia/lymphomas
   c. Reproduction in mice
5. The Loescher data are not replicable and are not a cancer model
6. The value of the Henhouse Project is speculative and the exposures are not relevant to the epidemiology data on cancer or miscarriages.

I am enclosing a copy of the relevant Japanese studies, as translated into English (I can also provide a copy of the original research reports in Japanese if this would be desirable). These are very well designed, conducted, and analysized, with a QA/AC program that is as good as the U.S. National Toxicology Program.

Chapter 7. Generic Issues on Epidemiological Evidence

Many of my comments above are relevant to this discussion.

Comments on Chapters 8 – 19

Chapter 8. Epidemiology of the Leukemias
Chapter 20. Dose Response Relationship

Dr. Sander Greenland will comment on these sections.

Chapter 9. Epidemiology of Adult Brain Cancer

Scientific evaluation to summarize a number research reports is much more sophisticated that presented here. Different studies are inherently more informative and should be accorded more weight. The Pro & Con presentations are overly simple. Too much weight is given to the epidemiology and too little weight is given to the relevant information from biophysics, experimental laboratory and whole animal bioassays. The results are inconsistent with the conclusions from those reached by the NIEHS, NRPB, and NIEHS.

Since Reviewer 1 has decided that 'Childhood Leukemia' is virtually certain to be related EMF exposure, that this then increases the strength of all the other evidence. There is no basis to support this kind of thinking.

Chapter 10. Childhood Brain Cancer

Scientific evaluation to summarize a number research reports is much more sophisticated that presented here. Different studies are inherently more informative and should be accorded more weight. The Pro & Con presentations are overly simple. Too much weight is given to the epidemiology and too little weight is given to the relevant information from biophysics, experimental laboratory and whole animal bioassays. The results are inconsistent with the conclusions from those reached by the NIEHS, NRPB, and NIEHS.

Since Reviewer 1 has decided that 'Childhood Leukemia' is virtually certain to be related EMF exposure, that this then increases the strength of all the other evidence. There is no basis to support this kind of thinking.

Chapter 11. Breast Cancer

Scientific evaluation to summarize a number research reports is much more sophisticated that presented here. Different studies are inherently more informative and should be accorded more weight. The Pro & Con presentations are overly simple. Too much weight is given to the epidemiology and too little weight is given to the relevant information from biophysics, experimental laboratory and whole animal bioassays. The results are inconsistent with the conclusions from those reached by the NIEHS, NRPB, and NIEHS.

Since Reviewer 1 has decided that 'Childhood Leukemia' is virtually certain to be related EMF exposure, that this then increases the strength of all the other evidence. There is no basis to support this kind of thinking.

Chapter 12. All Cancers

The U.S. occupational cohort studies are very strong in showing that there is no overall effect on cancer rates (Savitz et al.,; Sahl et al.,; Kelsh & Sahl)

Chapter 13. Miscarriage

The main weaknesses of the new studies (Lee et al., and Li et al.,) are that there is substantial evidence that there are no miscarriage effects from field exposures and the implausibility that any exposure over 8 mG would account for such strong effects.

You argue (Table 13.2.2) that (F.5.) "One should require some evidence for a specific bias before decreasing the degree of confidence because of bias." This perspective would be more valuable if the studies had been published to allow for appropriate peer-review.

By you discounting the relevance of the results from Schnorr et. al.,, are you suggesting that issues of the 'EMF mixture' are not relevant? Isn't this inconsistent with your approach?

Chapter 17. Heart Disease and EMF Exposure: Evidence

I am a co-author of one of the cited studies (Kelsh and Sahl, 1997). This literature is so new that it is not ready for 'risk assessment'. Weaknesses in the cited studies, the difficulty of using cardiovascular mortality assigned on death certificates as the outcome measure, selection factors into specific occupations and the potential for confounding should substantially dampen excitement in this area. In Kelsh and Sahl (1997), we performed an analysis comparing cardiovascular mortality between 'exposed occupations' and the general population (in Southern California). The SMR was no higher than .65 with the upper bound of the confidence interval at .77 (see Table VI, Kelsh and Sahl, 1997). Analyses of the Savitz results are similar. For our internal analysis, we used 'office workers' as the reference. Established differences in cigarette smoking, alcohol consumption, and diet are credible candidates for confounding. An increased level of work related exercise in the craft occupations would offset these. These factors may influence selection into the occupations with higher EMF exposure and influence length of service in these occupations. Another important consideration is that the overall rate of CVD mortality has substantially decreased, so that higher exposure scores are assigned to groups with higher background risk. The ratio 'Acute' and 'chronic' cardiovascular mortality has also changed over the last three decades, with both age and regional differences. Finally, the original hypothesis that increased decreased variability in 'intra-beat interval' has not held up in the laboratory.

That your team is willing to go so far with so little data is telling. The available data was enough for Reviewer 1 to conclude that he has a .45 'degree of confidence in causality' (with an upper bound of .70). How can this be given the available evidence?

Summary of my positions:

Key Themes Developed in The Written Comments

1. The authors fail to identify a specific exposure condition that is linked to specific disease. The reviews should be based on specific exposure-disease models to have scientific validity. Vague theories of causation are difficult to discredit.
2. There are logical errors; the most disconcerting is how the authors transform ignorance about health impacts into knowledge by assuming how the results from future research will support a causal mechanism;
3. No biologic model is described to support theories of causation.
4. Without clear definitions of the relevant dose, the 'patterns of evidence' for exposures cannot be evaluated.
5. Strong knowledge about biophysics does not support cancer initiation, promotion, or reproductive hazards.
6. Laboratory data do not support cancer initiation or promotion.
7. Whole animal bioassays do not support cancer initiation or promotion.
8. Recently published data on miscarriages is not consistent with the laboratory and animal bioassay data.
9. The epidemiological evidence has not been shown to be free of confounding or bias.
10. The conclusions of the authors are not consistent with the conclusions of independent expert panels, which used the same data.

Attachment B

Specific Questions That CDHS Has Requested Comment On
[Letter from Dr. Raymond Neutra, July 13, 2001]

1. We have taken the position that we are not greatly influenced by arguments based on physics and simplified biological models that suggested that residential and occupational levels of EMFs can't possibly produce bio effects. We say that theories should be used to predict results that are falsifiable and should not be used to discount evidence. Thus, our prior degree of confidence is not vanishing [ly?] small. Do you agree? Please comment.

   As supported in my main comments, you do not appreciate the value of the biophysical data to evaluate risks from a physical agent. You need to add expertise to your review team in order to correct the error in the draft.

2. Each of the three core reviewers have laid out their initial (prior) degree of confidence that residential or occupational EMFs could produce relative risks of various sizes. These estimates are constrained by what we know about animal bioassays for cancer and by the lack of dramatic change in disease rates after the introduction of electricity and as the use of electricity increased. Reasons are given for these judgments. Do they seem reasonable? How much higher or lower would your a priori degree of confidence be for any environmental/occupational agent? For EMFs? Why?

   I do not understand (even though I am very familiar with the relevant scientific literature), or agree with your judgments, especially those of Reviewer 1 and 3. Nor am I as excited by the miscarriage results, since no other literature supports this (and much of it discounts these results) and the 'story' does not make sense (that a one-time exposure above 8 mG would be this bioactive, or detectable given all of the other exposures that are commonly experienced). When I started work in this area in 1984, I was influenced by the work of Adey and his colleagues, Blackman and his colleagues, and the PMR studies on occupational groups. My initial prior would be around .25, and was increased by the Savitz and London results. EMF-RAPID failed to find experimental laboratory effects and the NTP bioassays were negative (Canadian, Japanese, and U.S.). Linet, McBride, and the English failed to support a childhood leukemia effect at 2 mG, but Greenland and Ahlbom found support for an effect at 3 – 4 mG when pooling (The Greenland and Ahlbom results do not impress me nearly as much as the three CDHS reviewers – I would put myself much closer to the 'discussion' in the manuscripts). The biophysics arguments got a lot stronger over the years, led by Weaver. Many tried to find a new biophysical mechanism, but they all failed. None of the experimental laboratory results withstood scientific scrutiny. My studies of electrical utility workers reassure me that the PMR studies were in error. The melatonin hypothesis hasn't gotten legs. The pattern has not gone well – better studies don't support the early work. The advocates increasingly rely on vague theories of causation and are unwilling to state clear exposure-disease models. The best scientists are leaving the field, and those who remain can't get funding from NIH, even thought there are has been $500 million in seed research funds. I am confused by the childhood leukemia pooled results, so am willing to stay open to possible effects. My posterior is about .15 (I could understand higher posterior's, but nothing like those expressed by the CDHS team).

3. We were not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects nor were we convinced of a chain of events that led to pathology. Yet we did not let this pull our degree of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment.

They tried very hard, yet were not able to find a mechanism. This should count for something.

4. We viewed the animal pathology literature as largely null, with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment.

   The animal studies are very relevant to the evaluation of risk and should caution your exuberance about the epidemiology. My comments in the main section support this.

5. Not all epidemiologists would agree with our position that relative risks between 1 and 2 should be taken seriously unless there is specified evidence for confounding or bias to explain it away. Do you agree? Please comment.

   It all depends on the strength of the available evidence. In this case, given the effort and the reassuring results from biophysics, experiments with cells and tissues, whole animal bioassay, and the epidemiology (which is not consistent on all stories), you should be very cautions about the epidemiology results.

6. We said that a lack of specificity in the association of EMFs with subtypes of cancer and evidence for effects on various types of disease did not pull down our degree of confidence and might even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature. Do you agree? Please comment.

7. Have we done an adequate job in presenting the arguments for and against causality or are we assigning weak arguments to the "con" or the "pro" position?

   Your pro arguments are speculative while the con arguments are based on observation. You consistently turn ' good ideas' into evidence to support causality.

Attachment A. An Example of the Importance of Wording: Opposition For Federal Funds For Stem Cell Research

Survey Organization: International Communications Research
Sponsored by the National Conference of Catholic Bishops

Questions: Stems cells are the basic cells from which all of a person's tissues and organs develop. Congress is considering whether to provide federal funding for experiments using stem cells from human embryos. The live embryos would be destroyed in their first week of development to obtain these cells. Do you support or oppose using your federal tax dollars for such experiments?

Support	24%
Oppose	70%
Don't Know	5%

Sample: 1,013 adults
Methodology: Telephone interview conducted June 1-5, 2001
Note: Percentages may not add up to 100% because of rounding

Survey Organization: ABC News
Sponsored by Beliefnet

Questions: Sometimes fertility clinics produce extra fertilized eggs, also known as embryos, that are not implanted in a woman's womb. These extra embryos are discarded, or couples can donate them for use in medical research called stem cell research. Some people support stem cell research, saying it's an important way to find treatments for many diseases. Other people oppose stem cell research, saying it's wrong to use any human embryos for research purposes. What about you—do you support or oppose stem cell research?

Support	58%
Oppose	30%
No Opinion	12%

Sample: 1,022 adults
Methodology: Telephone interview conducted June 20-24, 2001

Email: Jcollins@dhs.ca.gov

August 28, 2001

Jack Collins
California EMF Program
1515 Clay Street, Suite 1700
Oakland, CA 94612

Dear Mr. Collins:

I am writing to provide comments concerning two documents prepared by the California EMF Program: 1) *An evaluation of the possible risks from electric and magnetic fields (EMFs) from power lines, internal wiring, electrical occupations and appliances;* and 2) *policy options in the face of possible risk from power frequency electric and magnetic fields (EMF).* As you know, these are extensive documents and due to the limitations of time available for me to examine them, my comments are brief and focused, largely on the methodology used to develop the evaluation.

First, the California EMF Program is to be congratulated on attempting a review of this magnitude, while also attempting to apply new methodology for synthesizing information. The scientific community is repeatedly asked to summarize and synthesize information for the purpose of policy development. The approach used offers another potential option for deriving expert judgments.

With regard to the overall methodology, I have several comments:

- The methodology for eliciting expert judgment is novel in this application. Hence, there is inherent uncertainty associated with the report itself that extends beyond the judgments offered by the reviewers. We do not know the extent to which the review process itself is repeatable or inherently subject to bias, particularly from the reviewers selected. Of course, this limitation might be extended to any other expert synthesis, but the California EMF Program has introduced a new methodology that has not yet received application in this form.

- In this regard, the characteristics of the three reviewers may be critical. It is hard to know to what extent the reviewers and their characteristics influenced the ultimate judgments. Was there a representation of the involved scientific disciplines?

- The application of Bayesian methodology is novel, but dependent on the ability of the reviewers to specify priors that are not influenced by the controversy that has long surround EMF. I am skeptical as to whether priors could be truly specified that have not been influenced by the controversy surround EMF. To what extent are the judgments dependent on the prior?

- The report discusses some of the complexities of exposure estimation and the complexity of the EMF mixture. It could be strengthened by a stronger consideration of the likely degree of measurement error (probably quite substantial) and its implications (probably biasing strongly toward the null). Given that the proper biologic metric is uncertain and that misclassification is inevitable, the literature could be viewed as inherently “conservative” in identifying effects.

- The reviewers arrive at a judgment as to the probability of a causal association. This approach is reasonable and explained with appropriate cautions to readers of the report. Implicitly, smaller effects have a lesser probability to be identified as likely to be causal using this paradigm. Nonetheless, as a decision making tool, the approach has merit.

- The report places emphasis on case-control and cohort studies. Evaluation of disease rates for temporal trends consistent with the effects is also noted as one possible source of information. In fact, ecological studies may be a particularly informative source when exposures in case-control or cohort studies are poorly measured or relatively uniform. This may be the case in some of the populations studied.

- While I have not followed the literature on EMF closely, I judge the evidence tables to be quite comprehensive and well presented.

At this time, the document on policy options appears to be rather preliminary. I would hope that a subsequent version would more effectively link consideration of the options to the evidence evaluation.

I hope that these comments are of assistance as you evaluate and revise these important documents.

Sincerely,

Jonathan M. Samet, M.S., M.D.
Professor and Chair

:dk

Radiation Effects Department
National Radiological Protection Board
Chilton, Didcot
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UK

Tuesday, 04 September 2001

Evaluation of Possible Risks from EMFs (Draft 3)
California EMF Programme

Dear Professor Neutra

Thank you for inviting me to comment on the above draft document. At 300+ pages with extensive appendices this represents a substantial piece of work. I notice that you would like to receive all comments by September 10th and will therefore confine myself to addressing the general questions raised in your covering letter. I should point out that my background is essentially that of a biologist. Although, as secretary of the NRPB's AGNIR, chaired by Sir Richard Doll, I have been present when the EMF epidemiological evidence has been discussed, I have no professional expertise in this area. The opinions I venture below are very much my own, unless otherwise stated.

Q1 I think that, when making a judgement on the plausibility of an environmental agent acting as a possible carcinogen, you should take all the evidence into account and consider the physical plausibility, the strength of the biological evidence as well as the epidemiological evidence, particularly when relative risks are small and the possibility of selection bias, residual confounding and chance may provide an alternative explanation.

Q2 I would have taken a more critical view of the *a priori* degree of confidence that can be expressed with regard to the possibility of biologically significant interactions with environmental EMFs. The view that the very weak electric fields induced by these exposures, compared to endogenous electrical activity, might have significant effects and that we have had no time to evolve specific defence mechanisms (page 28) is somewhat speculative. The analogy with UVR (lines 33 and 34) is interesting. What one can say here is that, in contrast to EMF biology, there has been steady scientific progress in the understanding of UVR interactions with tissue and of the molecular basis for skin cancer induction and immune suppression over the last 10-20 years, even if its exact role in melanoma aetiology is not yet understood.

Q3 With regard to other known carcinogens for which supporting animal evidence was initially lacking, the large relative risks for smoking (page 8, line 51) and the existence of a clear dose-response relationship gave strong support even in the absence of these data. The difficulty I have is with the interpretation of small increases in relative risk in the absence of support from biological evidence. With regard to childhood leukaemias and EMF, AGNIR (2001) came to the conclusion that "In the absence of clear evidence of a carcinogenic effect in adults, or of a plausible explanation from experiments on animals or isolated cells, the epidemiological evidence is currently not strong enough to justify a firm conclusion that such fields cause leukaemia in children." We did add, however, "Unless, however, further research indicates that the finding is due to chance or some currently unrecognised artefact, the possibility remains that intense and prolonged exposures to magnetic fields can increase the risk of leukaemia in children."

Q4 I don't really see how a largely null animal pathology literature can increase confidence in the epidemiological literature. Experiments should be designed to tease out the biologically effective exposures, testing falsifiable hypotheses and maximising the chance of tracking down the aetiology of an effect. Sometimes, good animal models are lacking, or inappropriate exposures carried out. But I think that it would be wrong to conclude that the epidemiological evidence is strengthened on the grounds that the animal data were negative. To assume the existence of an appropriate environmental 'mix' that is more biologically effective than experimental exposures begs the question.

Q5 As for Q3.

Q6 I would have thought that generally, specific agents are associated with specific types of disease for mechanistic and biological reasons. It is, however, easy to understand why different risk factors and diseases (but I think of largely viral origin) might be associated with something that depresses immune system responsiveness, such as AIDs (page 69), or immune suppression in transplant patients to give another example. I would suggest that experimental evidence for EMF effects on immune responsiveness is pretty weak. I think that it is wrong to conclude that the lack of association of EMFs with different subtypes of cancer increases confidence in epidemiological associations between disease X and EMFs.

Q7 I would have taken a more critical view of the quality of some of the biological studies. In particular, the life-time animal studies and those on transgenic animals under the EMF Rapid programme were of really impressive quality; some of the others, which appear to have been given equal weight, were of a somewhat lower quality.

Q8 Perhaps for the confidence range 10-50% you could have 'less possible' and for 50-90% have 'more possible' rather than just 'possible' for both.

I hope that this is helpful.

Yours sincerely

Rick Saunders

cc Dr R Cox

Ref: NRPB. ELF Electromagnetic Fields and the Risk of Cancer. Report of an Advisory Group on Non-ionising Radiation. Docs 12(1), Chilton, NRPB (2001).

Comments from David Savitz

Thank you for the opportunity to comment on the draft of the EMF report. I will respond to the specific points raised in the letter of July 9, 2001 to reviewers, as well as commenting on other issues that these points do not directly touch on. This is a remarkably thorough effort to come to grips with a large body of literature and bring it to bear on policy, which really is admirable, even if it exposes real limitations in the underlying knowledge base and the methods for using it.

Response to specific questions:

1. I agree that theoretical arguments do not explain away data, but I would approach the overall assessment of causality by integrating the epidemiologic evidence (with its strengths and limitations) with the experimental and theoretical arguments from other disciplines into an integrated evaluation. If the epidemiologic evidence were strong enough, the "hit" from theoretical counterarguments would be of little importance, whereas when the epidemiologic evidence is modest at best, an integrated assessment of the evidence would in fact give a fair amount of *relative* weight to the theories from physics. I also view the other disciplines not just as help in interpreting the epidemiology but also rather as independent lines of evidence, which, along with epidemiology, help to make an overall judgment.

2. The uncertainty that exists at present makes a rather wide range of priors reasonable, and those of the three reviewers certainly fall into that range. They are probably giving more credence to the possibility than most of those who look at this issue would (though I don't have empirical evidence to support that contention). I would incorporate the physics, biophysics, and experimental evidence into my prior and say "not very likely" to all health effects, with the epidemiologic evidence modifying that prior to varying degrees across the various outcomes.

3 & 4. See points above.

5. This is a key point – the lengthy, detailed consideration given to "what explains the positive epidemiologic findings" seems in my view to overstate the strength of findings that need to be explained. That is, with weak associations, inconsistent in magnitude, there's not a great need to invoke deterministic explanations based on causality or bias. Modest evidence for each or the relative merits of causality versus bias presuppose something about the strength of association to be explained.

6. Specificity or lack of specificity is not a major concern, except insofar as findings for one outcome help to form insights that are applicable to other outcomes.

7. The length and detail of the arguments is far beyond anything done before, and may well be more than is needed or helpful. It is hard for even those with a real academic interest to work through all the details, and it is unclear how much of a target audience there is for this level of detail.

8. I think that the phrasing and numbers are fine for clear communication purposes.

Other issues:

1. The conclusions from this report, the posterior assessments, clearly lean further in the direction of believing that EMF poses a hazard than you would find from a randomly selected group of scientists or policy makers who follow this issue, in my opinion. The question comes up as to whether this is due to the priors of the three individuals, the systematic examination assigning more confidence to the possibility than one gets with pure intuition, or some bias or at least unusual aspects of the way that the likelihood factors were quantified. I really do take seriously the idea that when one dissects this out in the extreme detail that you have for this report, conclusions differ from those we would obtain using intuition. However, the burden of proof on bias versus causality noted above seems to start with the

premise that an association is really present. If it's there at all, it's weak and imprecise, and surely that should get factored in somehow as well.

2. The confidence placed in the two as yet unpublished studies on spontaneous abortion sponsored by the California EMF program seems far out of balance with the quality or confidence in the research. There are a few issues there – the totality of the evidence, not just two new studies; the assessment independent of who funded or encouraged the research; and the lack of opportunity for the scientific community to make an informed judgment of its value. Going from new data to summary assessment is unwarranted in my view.

Comments: Joachim Schüz, University of Mainz
(Phone: +49 6131 17 31 13, Fax: +49 6131 17 2968, e-mail: schuez@imsd.uni-mainz.de)

Reviewer-Questions:

1.
I fully agree with you that these theories should not discount epidemiological evidence. When it comes to causality, however, the lack of a biological mechanism is an important issue.

2.
No comment. Actually, the part about the a priori confidence was the part I liked least in your report. But maybe I didn't understand the rational of it.

3./4.
Once again, I think that the lack of mechanistic or animal data on adverse effects of EMF does not pull the degree of confidence in the epidemiological data down. It may be possible that the true mechanisms are more complex than those that we have examined so far. I'm not very convinced that one of the current hypothesis is actually the true mechanism, neither the melatonin-hypothesis, the contact current-hypothesis, or the transients-hypothesis. Nevertheless, epidemiology is only one piece of the puzzle. Moreover, it is not the case that the results of these epidemiological studies are irrefutable. It is also not the case that there is a lack of experimental evidence because they were no experiments; there were numerous studies on effects and mechanisms, but as you correctly said, the results were largely null. So I still think this decreases the degree of confidence in causality.

5.
I agree with you that also small relative risks between 1 and 2 should be taken very seriously, if they arise from high-quality epidemiological studies. Nevertheless, one should be very cautious regarding bias when the risk increases are only moderate, and higher relative risks may be more convincing. For residential EMFs, one has to be especially cautious, because in most studies there are not only weak associations, but also the prevalences of exposure are very low (decreases the power of the study), the response rates are at most good-to-fair (selection bias is probable), and one looks at diseases for which little is know about the etiology (no idea about confounding or co-carcinogenity).

6.
I agree that a lack of specificity should not pull down the degree of confidence in this field. The reason is that in most studies the exposure assessment relies on measures that may be biased by non-differential misclassification rather than differential misclassification. Lack of specificity may be an important issue in studies relying on recall by interviewees.
However, it seems to me that there is some specificity in the association of EMFs with different types of cancer. To my mind, the childhood leukemia studies are much more convincing than studies for any other type of cancer. For adults, there also seem to be the strongest effects for hematological malignancies. I'm not convinced by the studies on brain cancer or breast cancer.

7.
I like the style of your presentation of the arguments very much. However, sometimes I got the impression that some of the arguments rely too much on single studies, and sometimes not even the best studies (one example is (F2) in Table 8.2.2: the impact of selection bias should be discussed in the light of large studies that materially contribute to the EMF-leukemia-association, particularly the Linet study; e.g., the case-specular method for the Savitz and London study is completely irrelevant for the interpretation of the meta-analysis finding at 0.4μT by Ahlbom – and the studies that were part of this meaningful analysis that were affected by selection bias all show selection in the same direction).

8.

No comment. I felt that your phrasing was ok.

Chapter 8: Leukemia

Page#	Table#	Line# or Comment# in Table	Comments
75	8.1.2	entire Table	This Table brings no new information compared to Tables 8.1.3 and 8.1.5; in the opposite, it is very confusing that the ORs are different from the originally published results and it is not obvious for the reader where this difference comes from. Suggestion: Delete Table 8.1.2
89	8.2.2	(A7) / (F3)	As shown by both meta-analysis from Greenland and Ahlbom, the McBride study is homogenous to all other studies; the reason why the Green study is different from all other studies is poor quality rather than bias. Suggestion: Delete (A7) and (F3)
89	8.2.2	(C1)	This is not a good argument; consistency could result from a consistent type of bias. Suggestion: Delete C1
89	8.2.2	(C2)	This is not entirely true. Nondifferential misclassification may inflate risk estimates for intermediate categories. This may be an explanation, why in some occupational studies the odds ratios for intermediate exposure categories are higher than for the highest exposure category. Suggestion: Modify C2
89	8.2.2	(C3)	Suggestion: There is some evidence that an inflation of the risk estimates is common to those studies that show the most convincing associations (i.e., the long-term measurement studies), which is attributable to differential nonparticipation. It is unlikely, however, that the entire association can be explained by selection bias.
90	8.2.2	(F7)	A) There is no need for a consistent upward bias across <u>all</u> studies – the question is whether there is an upward bias in studies that contribute to the leukemia excess with higher MF; thus, particular attention has to be paid to the Linet study (and for this study, overestimation of ORs from nonparticipation has been shown (Hatch et al., 2000)), and to the measurement studies that have similar or even lower participation rates (Canada, Germany, UK). B) For the total population, childhood AL may be not related to SES, however, it is in the EMF studies; that's because case participation is not related to SES, but control participation (differential selection bias). C) One argument for selection bias: the low response rates of the measurement studies. D) One argument against selection bias: the association reported from the Scandinavian studies, where, because of MF estimations, nonparticipation was no issue. Suggestion: Modify top 1 with B, add C and D
91	8.2.3	(F3)	This is only one study. There are other studies that show no association between traffic fumes and childhood AL (e.g.,

			Raschou-Nielsen et al., 2001). Suggestion: Delete F3; modify F2 by mentioning that controlling for traffic density had no effect in the meta-analyses
93	8.2.5	(F1) / (F2) / (C3)	This is not very convincing, because it doesn't take into account the strength of assocation. If selection bias inflates all risk estimates slightly, then one would expect to see the majority of risk estimates above the null. Suggestion: A more convincing argument for consistency are the negative tests for heterogeneity of the meta-analysis. (see 8.2.6, F1)
94	8.2.6	(A1)	Suggestion: Of the wire code studies, the largest one shows no risk whatsoever.
97	8.2.8	(A2) / (F2) / (C2)	I wonder if it is necessary to highlight the observation that in the Swedish study the association was stronger when the analysis was restricted to single-family homes. Even if this is not attributable to misclassification, it could be explained by random variability because there is only a handful of exposed cases in the Swedish study. Suggestion: Delete A2, F2, C2
97	8.2.8	(C1)	The strongest argument against A1 is that, because the attributable risk was estimated to be 3-4%, a disease excess can be hardly demonstrated by incidence data. Suggestion: Add this to C1
99	8.2.15	Bias not proven Experimental evidence	I think that it is justified to say that it ➢ Pulls down confidence quite a bit. based on the modifications for Table 8.2.2 I think that it is justified to say that it ➢ No effect or decreases confidence somewhat. There are only few areas of environmental hazards where there has been such an extensive research on mechanisms. And nothing convincing has been found.
General remark #1			Generally, it would be better not to weigh all the studies as if they were of the same quality. For some aspects it would be more appropriate to discuss them in the light of the high-quality studies (e.g., the small Green study is mentioned a couple of times in the Tables, while the Linet study is cited only once).
General remark #2			I think it would be worth the effort to prepare an appendix with some new papers that are very supportive of the overall outcome of your evaluation: * Ahlbom et al. (2000): The meta-analysis of the studies with an improved design shows a significant association, based on 44 observed cases versus 24 expected * Schüz et al. (2001): The new German study is the first to show a dose-response-relationship, for an exposure measure that is probably less affected by nondifferential misclassification (night-time exposure) * Pfluger and Minder (2001): The Swiss railway worker study is quite convincing, because it comprises groups of workers that are comparable except huge differences in MF exposures, and an elevated risk was seen for those with the highest exposures

Chapter 10: Childhood Brain Cancer

Page#	Table#	Line# or Comment# in Table	Comments
134	10.1.2 (figure)	Verkasalo (1993)	Suggestion: There should be a footnote explaining that one exposed case had three primary brain tumors; thus, the number of exposed subjects with brain cancer is three instead of five (and the respective odds ratio would be considerably lower).
139	10.2.5	(F3)	This argument is not very convincing. I have no idea about one single potential risk factor for childhood brain cancer for which one can rule out an OR of 1.2 with a wide confidence interval. Suggestion: Delete F3.
140	10.2.8	(C1) / (C3)	It should be emphasized that (F1) has nothing to do with MF.

Asher Sheppard Consulting

Asher R. Sheppard, PhD Consultant in Environmental Science

September 9, 2001

Mr. Jack Collins
Division of Environmental and Occupational Disease Control
Department of Health Services
1515 Clay Street, Ste. 1701
Oakland, CA 94612 By e-mail: jcollins@dhs.ca.gov

Re: EMF Risk Evaluation and Policy Analysis

Dear Mr. Collins:

I wish to thank Dr. Neutra for the opportunity to review and comment on the Risk Evaluation and Policy Options documents concerning exposures to power frequency electric and magnetic fields. I offer my comments with the caveat that I was not able to delve deeply into the details of information on individual studies and specific statements on studies or groups of studies and was not able to give the time needed to make a thorough review of many interesting topics. I hope my comments have some value despite these limitations.

I found the Risk Evaluation and Policy Options exceptionally transparent approaches to summarizing information over a large number of scientific disciplines and with respect to numerous societal concerns and perspectives. These societal perspectives were neatly captured by the four social models addressed in the report (utilitarian, social justice, risk certainty, and non-interventionist). I thought this transparency was aided significantly by the tabular presentation of interpretations of specific research studies and topics. I recognize the efforts taken to assemble the information and to provide specific judgments together with the values underlying them. This gives this document a clarity and interpretive richness that is not often found in critical scientific literature reviews.

I thought the Bayesian approach was carried out quite successfully and appreciated the opportunity to observe how scientific judgments could be made with this approach. Although not enamored of the Bayesian approach, I thought it had great didactic value and met the goal of serving heuristic purposes and needs of the policy analysis. This approach was a major factor in the transparency I referred to, but the value of the report was severely hindered by the fact that only three people conducted the exercise and more so, in my reading, by the fact that I am familiar with each of them. I do not imply disrespect for the knowledge and independence of each of the three staff scientists, but found myself often wondering how this exercise might have fared with a broader population of scientists and regulators. I also found myself identifying particular perspectives with what I believe to be the scientific personalities of the three, rather than accepting them impersonally.

I am not persuaded by the risk evaluation exercise that the numerical flavor given by assigning probabilities is useful. I remain very uncomfortable using the tools of statistics to assign numerical probabilities to causal relationships. For one, numbers give a false note of definiteness to matters that, in the end, remain very indefinite. Knowing that someone feels a relationship is 65% likely and 35% unlikely is, for me, not knowing very much when what I need to know is essentially binary information, that is, whether there is a causal relation or not. Assigning 1 and 0 is vastly different from permitting all values between 100 and 0 to assess whether the true assignment is 1 or 0. I was glad to see that one of the messages of the document is that continued scientific research is justified because only continued application of the scientific method can address whether there is or is not a causal relationship between EMF exposure and a disease endpoint. For another reason, I wonder about the stability of such probabilities that may, for example, be strongly affected by purely psychological factors such as consonance or dissonance with prior beliefs or by the a psychological shock factor if the latest data or new reading of the literature causes an, “Ah ha!” reaction. In

summary, I found the documents flawed to the degree that probabilistic statements, which I believe are largely guesses in statistical dress, appear as substitutes for statements rooted in scientific and statistical fact. On the other hand, I find considerable value in the openness of this risk evaluation in contrast to expert evaluations where guesses can be clothed in other ways.

Despite recognition of the four social models, the policy analysis is most influenced by the utilitarian model. This is perhaps inescapable when dealing with electricity, which is an economic factor of great significance and universal prevalence in our society. I think it commendable to have tried to make balanced statements, but in the end avoidable deaths and dollars still jump out as the most salient information.

The documents are a rich resource for continuing discussion on each of the many topics broken out for evaluation. I thought successful and defensible balances were struck between the choices of narrowing viewpoints to isolate nearly identical endpoints and broadening them to give perspective on an area of physiological or epidemiologic research. This comment applies not only to the groupings of studies but also to the topics used in the "Against Causality" and "For Causality" entries.

Although well organized and despite the introductory material, I sometimes found myself lost when looking through the report. Some flying headers (in addition to the footers (that I only noticed midway through!) could help. The horizontal layout for the small font size of the text was difficult to read and scan. Another visual handicap for me was that the two columns seemed rather wide for the font size and line spacing.

In reply to Dr. Neutra's eight specific questions:

1) I am not in full agreement. In abstract terms, experimental data are more valuable evidence than theoretical analyses. However, where the data are highly uncertain, the question of whether to trust shadowy data can be helpfully answered by use of theory. If, as in the noise-based analyses of weak EMF fields, theory shows a gap of several orders of magnitude between the EMF level believed to be effective in some research study and a theoretical minimum, I find the theoretical analysis is a powerful argument against weak data. The frequently made appeal to unknown (and perhaps unknowable) factors attributed to biological complexity overlooks the very power of the biophysical argument, which is its simplicity. Specifically, there has to be some first interaction of a field with charges or moving charges and the magnitude of this interaction can be estimated without a sophisticated biophysical model. *The power of the biophysics argument is its simplicity and this simplicity is not a weakness as portrayed in the Risk Evaluation*. I acknowledge that any simple model may make a false assumption or overlook a key element. One such assumption may be equating 0.3 or 0.4 μT with the effective field in epidemiologic studies, when the effective field may be some other exposure feature highly correlated with exposures in the tail of the distribution function. Once the margin between theory and experiment or epidemiology is reduced to a small factor, then questions of biological complexity become relevant and much more interesting. In my opinion, there has been a degree of fuzzy thinking about the complexity of biological systems as if complexity were an answer to fundamental questions of information theory and energetics. Once the biological system receives a signal, biological complexity may be very significant, but biological transducers in the eye, ear, skin, and elsewhere are devices for movement of ions in a cell. It makes sense to examine mechanisms involving charge movement when investigating for a first cause of field interactions and to use simple models to do so.

2) The priors given on p 29-30 (sec. 2.4) are reasonable and well argued, but unpersuasive individually and collectively. I was not swayed by the thought that environmental novelty and non-natural origins heightened the probability of harmfulness. The bounds chosen for undetectability and easy detectability of EMFs as harmful agents were reasonable. The argument (Researcher Three) that the EMF mixture might convey greater risk than an agent with one characteristic was not persuasive because it made the unfounded assumption of a probable risk from multiple adverse interactions.

I mistrusted that these are indeed a priori judgments. Each of the three DHS scientists, and particularly Drs. Neutra and DelPizzo, has been prominent in the EMF research community for many years and has been weighing the pros and cons of EMF research continuously. How could they presume to have truly prior views, or did I miss something? As noted before, I am not fond of throwing probabilities about when they are essentially guesses and that applies here too.

3) When considering epidemiologic data, I agree that the lack of a biological mechanism is not a strong reason to discount the data. The absence of mechanism is good reason to check and check again, to look for confounders, biases, and so forth, as has happened for the childhood leukemia data and to lesser extents for other endpoints. While not ignorable, it is not a factor that can prevail against a large, thoroughly examined, set of data obtained with reasonably good techniques and decent exposure assessment.

4) For much the same reason as above, the absence of strong evidence for animal pathology, while not ignorable, does not strongly affect causal inferences drawn from the epidemiology data. The fact of some positive data that have not been resolved (particularly mammary cancer in rats), inability to conduct studies at very high doses, and the limited statistical power of animal studies, augment my belief that the mostly negative animal pathology data are insufficient reason to significantly discount the epidemiology findings.

5) Context is missing here and would influence the answer. Do you mean a case-control study? a large cohort study? a meta-analysis? To this non-epidemiologist, the dividing line here seems to be between those whose experience and observation of history show that small risk ratios rarely mature into confirmed causal relationships, and those who look at the data in isolation of history and focus on statistical features and study quality, among many other factors. I am sympathetic to the prejudice that arises from historical experience, but believe that such prejudices should be put aside until research on a topic has matured, for example as indicated when there are enough studies to do meta-analyses with fairly large numbers across the exposure distribution. I strongly favor letting the statistics tell the story even if the apparent RR is below 2.

6) If EMFs are causally related to leukemia, brain cancer, and particularly subtypes of these cancers, we are so far from understanding the mechanism that I suspend any judgments that might be made from experience with chemicals and ionizing radiation. That is, the lack of specificity may be a feature of a hypothetical EMF mechanism about which we know so little that previous carcinogenic mechanisms are of dubious instructional value. However, from the pathologist's perspective on development of certain tumor types, this could be a facile and unintelligent remark. Therefore, dependent on details of the pathological nature of various tumor subtypes, the lack of specificity in cancer types generally is a weak argument for mistrusting epidemiologic findings. When applying this statement to EMF epidemiology, there is too little known about occurrence of cancer subtypes to make much use of pathology-derived views on etiology and therefore the apparent lack of tumor specificity is a still weaker argument against epidemiologic findings.

7) I have not evaluated the "pro" and "con" positions in depth nor have I read all with care, but my impression is that there were no evident biases. There were, however, consistent views evident in the "comment and summary" statements drawn from the contrast of "pro" v "con".
8) As noted above, I do not favor marrying numbers with seeming statistical precision to qualitative language when only qualitative terminology is justified. I recognize that the needs of the decision analysis drove an interest in producing numbers. The words used in the current phrases are in most cases just coded restatements of the numbers shown in the "confidence range" column. I offer as substitutes for the ranking of risks without an association with numerical values the following terms that, to the best of my ability, do not connote statistical concepts: *"Definite"; "Expected"; "Unexpected; "Remote";* and *"No Appreciable Risk"*. I would be just as happy to see only three values: *"Expected"; "Unexpected";* and *"Remote"*, because these should cover almost every realistic risk scenario. For example, a lifetime of heavy tobacco use is "expected" to cause a disease, but even adding up lung cancer, heart disease, pancreatic cancer, stroke, etc., it is not

"Definite". Some high asbestos exposures could be a counter-example, but I think the three terms *"Expected"; "Unexpected";* and *"Remote"*, capture most risks. One nice thing about having three categories is that it defies the split between greater than 50% or less than 50% that some segments of society put so much emphasis on.

Sincerely, Asher R. Sheppard

Comments on EMF Risk Evaluation

For Dr. Raymond Neutra
Cal HHS Agency
Tel 510-622-4900

I have carefully read the EMF Risk Evaluation and Policy Options Report.. Overall, it is clearly written, balanced, and temperate in its assessments and conclusions. Specific issues are noted on the attached page of notes.

In general, I have the following concerns:

1. While I support. the use of a Bayesian framework upon which to rest explanations of expert judgement, I wonder if this is sufficiently clear to the lay leader. Moreover, Bayesian analysis is not explicitly used in terms of computing likelihood estimates, but rather only invoked as a sort of philosophical approach. This is a bit confusing.

2. In the tables and text, the conclusions of various studies are cited as stated by the authors. Yet there have been numerous critiques of some of these papers for unsupported conclusions by their own data. This should be discussed.

3. Insufficient weight is given to the NTP studies. Failure to find any indication of carcinogenecity (not simply tumors) is very significant. Moreover,the lack of mechanistic data in studies explicitly designed to test cellular and. molecular responses is also significant. Tumors reported without histopath cannot be accepted as malignancies. The Soviet papers have insufficient detail to be evaluated.

4- It is not clearly argued, that th.e evidence supports association with Lou Celirigi disease, even at the level of "possible".

5. More discussion of exposure is appropriate, particularly if peak exposure is more significant than steady "background" - peak or variable exposures probably describe most occupational studies, and are more plausible in the eye of physicists.

Ellen Silbergeld
University of Maryland Medical School
10 South Pine Street
Baltimore, MD 21210
(written in Norway at a WHO meeting)

Other Comments**:**
Page **5,** line 9 -'Total miscarriages are estimated by Wilcox et al to be as high as 60-80% of all, conceptions. Depends on timing. Do we know about EMF**?**

Page 6, line 48-59 - Doesn't really do justice to physics argument**.**

Page 21, line 25 -1 like the Bayseian approach. Do you think all reviewers understood it?

Page 31-3 1, table 3. 1.1 - Presume 4 = check mark and x = x?

In the next 3 comments did you review the, papers or just take conclusions as valid? .

Page 34, table 4.1.1 - Biochem processes are not "random".
Page 46, table 6.1.10 - Why is this referred. to as, "stress preterms"? Were HSPs measured?
Page 47, table 6.1.11 - Same comment - prolif is not equal to OPC activity palpation is NOT a reliable measure of tumorgenecity. Why no histo?

Page 59, table 6.2.6, line A6 - Which. studies seem to have been interaction studies; EM Falone? (on balance animal data are unconvincing)
Page 74, table 8.1.1 – Nice to have size of study in table!
Page 80, table 8.1.4 - Most OCS studies had other exposures (see some of the papers)
Page 88, table 8.2.1 - Conclusion does not seem warranted
Page 99, table 8.2.15 - There is is no evidence +l-re "plausibility"
Page 205, table 9.2.2 - Breast cancer studies seem. stronger than Reutemu
Page 194, table 13.2.1 - Other stresses assoc with- prolonged VPT use
Page 207, table 13.3.2 - Agree that SAB risks, if;real, may well be related to peak expoconsistent with chemicals that increase SAB.
Page 226, table 14.2.12 - Temporality issues are different for developing toxicity; can't refer to "generic" issues.

Is there an overall problem in RR/OR - if general population is also exposed, who are the referents?

September 5, 2001

Dr. Raymond Neutra
California EMF Program
1515 Clay Street, Suite 1701
Oakland, California 94612

Dear Dr. Neutra:

I am writing to provide public comments on the California Department of Health Services (CDHS) *draft* report entitled, "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances." The views expressed in this letter are solely my own and do not necessarily reflect the opinions or beliefs of the organizations that funded this work. Funding was provided by the following electric utilities: Southern California Edison, Pacific Gas & Electric, Sierra Pacific Power Company, Los Angeles Department of Water and Power, Sacramento Municipal Utility District and San Diego Gas & Electric Company.

The focus of my comments is on the risk assessment methodology. My CV is attached for your review. I appreciate the opportunity to provide these comments.

Sincerely,

Abe Silvers

California Department of Health Services

"An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs)

From Power Lines, Internal Wiring, Electrical Occupations and Appliances"

Draft 3, April 2001

I have one key point. The methods presented in its current form are not useful for performing scientific risk assessments. My recommendation is for the CDHS to either use established risk assessment methods or to use formal (and quantitative) Bayesian methodology. In addition, I recommend that the number of authors be increased by including scientists with expertise in the disciplines that are relevant to the available scientific information. This would help to achieve authors who are more representative of the wider scientific community and to improve the relative weighting of data from the various scientific disciplines (e.g., laboratory experiments, whole animal bioassay, epidemiology, and biophysics). It would also be valuable to disentangle the 'risk assessment' from the 'policy evaluation' by using different teams with appropriate expertise for each task. I elaborate on these points below.

INTRODUCTION

A California Department of Health Services (CDHS) analysis of the potential association of EMF exposure to various diseases was submitted for public comment. The CDHS EMF Risk Assessment uses new methodological analysis for summarizing the results of studies about the relationship of EMF exposure to a number of disease categories. As is true for formal risk assessments, the available scientific information comes from many disciplines, in this case, four different disciplines. The disciplines are biophysics, cell biology, whole animal studies and human studies. The exposures are to extremely low frequency electric and magnetic fields present in residential, community and occupational environments. The diseases considered include several kinds of cancer, heart disease, and reproductive effects. Formal risk assessments using established methodology are difficult because of uncertainties and the new methods used by CDHS have introduced new potentials for error. The authors report that they encountered various difficulties in their attempt to provide a uniform summary of evidence. Different disciplines provide information on different levels of the human organism and integration of these levels may not be easily done.

The problems of performing a risk assessment were acute since the available data are inconsistent within the disciplines and between the disciplines. Meta-Analysis may not be sufficiently sensitive to summarize all the evidence and provided a low relative cancer risk that may be of the order of 1.5.

The CDHS attempted a numerical analysis based on a weight of evidence approach using Bayesian methods. Employing a weight of evidence analysis has always been a priority of the U.S. Environmental Protection Agency (US EPA). Recently, the U.S. EPA has produced guidelines for the risk of cancer based on the weight of evidence in animal studies (EPA). Their position is that primary evidence be supported by secondary evidence. This secondary evidence (in their consideration, pharmacokinetics and pharmacodynamics) should be consistent with results from whole body animal studies and results from epidemiological studies of human populations. The U.S. EPA risk assessment guidelines support the concept that evidence consistent with human physiology enhances the conclusions in a risk analysis. Epidemiological guidelines are not yet available.

EMF evidence derived from epidemiological studies is weighted with evidence from the other disciplines. The report devotes considerable effort to the pros and cons of this evidence. However, to summarize the evidence, the report embarks on the beginnings of a Bayesian analysis to arrive at a probability of confidence that EMF exposure causes the occurrence of a disease.

The basic Bayesian philosophy is to update the probabilities by introducing new evidence. If one assumes a prior likelihood based on historical evidence, then the likelihood can be updated with more evidence; therefore achieving a posterior likelihood that, due to exposure, the disease will occur. The method is based on using expert subjective opinion, updating their estimate of confidence and then summarizing their confidence levels into a uniform confidence level or range of levels. The authors, however, did not engage in a full Bayesian network analysis. The authors were told the subjective numbers may be not well received by the scientific community, and that the more intensive Bayesian analysis may not be understood by the scientific community. In a later section, we will discuss the limitations of the report's implementation of a Bayesian analysis.

BACKGROUND

The method used in the report is primarily predicated on two methodological developments, 'applied decision analysis' and 'medical decision making', which incorporates applied decision analysis. Applied decision analysis was given its impetus at Harvard University through the work of Raiffa and Keeny (Keeny and Raiffa) and at a later period at Stanford University through the Engineering Economics Department guided by Ronald Howard (Howard).

The methodology is based on subjective probabilities and has been successfully used in business decisions, in economic analysis, in medicine to minimize costs and for remediation strategies, and in risk analysis. The goal is is to improve policy decisions by improving on returns-on-investment. The method has also been valuable with respect to deciding on remediation strategies, such as for clean-up options of hazardous waste sites.

A major improvement in 'applied decision analysis' was provided by D. M. Eddy's work on the confidence profile method (Eddy). This method claims to take into account the bias in individual studies as well as uncertainty in the estimates of individual study parameters. There are considerable examples of the uses of applied decision analysis in deciding treatment strategies for a number of diseases. All these methods invoke the concept of Bayesian networks (tree structures) that incorporate subjective probabilities. The authors of the CDHS EMF report based their approach on an analogous method used in medical decision making for determining the predictive values of a medical test. These "sensitivity" and "specificity" procedures use likelihood odds to determine whether a test predicts if a medical problem is likely. Based on a priori pretest odds for a medical disorder, posttest odds for the disorder can be calculated with the following formula:

Pretest odds for A medical problem	$\times$	the likelihood ratio for the diagnostic test result	=	the posttest odds for the target disorder

For example, if you assume 50/50 chance or an odds of 1:1 of occurrence of a heart attack on a work-up on a man with chest pain, and if a test result gives the likelihood odds of 4.4, then applying the formula $1:1 \times 4.4 = 4:4$, an odds which is equivalent to a probability of 81% of a heart attack. The 4.4 is derived as shown in Table 1.

Table 1. Likelihood ratios for several levels of a diagnostic test result

		Myocardial infarction				
		Present		Absent		
		Number	Proportion	Number	Proportion	Likelihood ratio
CK Test result	≥ 280	97	$\frac{97}{230} = 0.4217$	1	$\frac{1}{130} = 0.0077$	$\frac{0.4217}{0.0077} = 55$
	80-279	118	$\frac{118}{230} = 0.5130$	15	$\frac{15}{130} = 0.1154$	$\frac{0.5130}{0.1154} = 4.4$
	40-79	13	$\frac{13}{230} = 0.0565$	26	$\frac{26}{130} = 0.2000$	$\frac{0.0565}{0.2000} = 0.3$
	1-39	2	$\frac{2}{230} = 0.0087$	88	$\frac{88}{130} = 0.6769$	$\frac{0.0087}{0.6769} = 0.01$
		230		130		

This example is from D. Sachett et al (Sachett et al). Thus new data changes the *a prior* odds to *a posterior* odds, i.e., from 1:1 to 4:4 or a 50% chance of having a heart attack to an 80% chance of having a heart attack. In this case, the initial odds are based on a subjective probability or confidence level, and the new data modifies this subjective probability. 81% is derived from $\frac{Posttest\ odds}{1 + Posttest\ odds} = \frac{4:4}{5:4}$. Conversely, the odds can be derived from $\frac{probability}{1 - probability}$.

CDHS BAYESIAN EVALUATION

To begin to resolve the issues raised in the introduction, a preliminary Bayesian analysis was conducted using "expert" opinion. The essence of the analysis was that after an extensive discussion of the evidence in the four disciplines, the experts would derive a confidence level expressed as a percent that is equivalent to the International Agency for Research on Cancer (IARC) criteria for weight of evidence as applied to cancer. The authors applied a probability level to each component of the IARC criteria. An initial level of confidence is derived using a series of questions that documents the level of understanding prior to the subsequent studies on EMF. They had further

discussions on how each type of evidence in one discipline correlates with the evidence in higher biological levels, culminating in its effects on the human organism. Each stream of evidence will have a structured set of questions to help answer whether the stream is uninformative, strengthening or weakening, predominantly strengthening or weakening. To express quantitatively a change in confidence brought about by reviewing the evidence, a Bayesian approach predicated on the medical decision model is invoked. It considers (Probabilities (P) and odds)

a) Prior odds: $\frac{P(cause)}{P(not\ cause)}$

b) Relative likelihood: $\frac{P(evidence/cause)}{P(evidence/not\ cause)}$

c) Posterior odds: $\frac{P(cause/evidence)}{P(not\ cause/evidence)}$,

where / denotes the logical phrase 'given'.

Bayes theorem stipulates:

$$\text{Posterior odds} = \text{relative likelihood} \times \text{prior odds as discussed in the section of medical decision making}$$

As stated in the introduction, the CDHS authors reviewed the available scientific literature and developed a series of pro and con arguments about the implied 'strength' of the evidence. This characterization of the evidence attempted to arrive at a degree of confidence of causality, using the IARC levels as a guide. The degree of confidence is depicted for each expert.

LIMITATIONS OF METHODOLOGY

The authors initially stipulated that there were constraints on the depth of the analysis. They could not use a more formidable model formally invoking Bayesian networks because they thought that the scientific audience would not accept the numbers with the model. It was also assumed that the audience would not understand the procedure

sufficiently to be able to evaluate the method and results. Thus a simpler methodology was utilized. This simplicity, in itself, generates many concerns about the procedure.

To begin, the authors used the IARC guidelines as a guide to generate probabilities. The IARC guidelines were not developed for this purpose, and the probabilities associated with the components of the guidelines do not have any scientific basis. They appear to be the subjective interpretations of the authors. In fact, 95% confidence could be 85% confidence. There is no actual support or evidence provided to suggest what the value may be. The main point is that there isn't any uncertainty associated with these numbers, and it is not clear what range of uncertainty is associated with each component. Further, it is not clear what uncertainty each expert has with respect to his or her own confidence level. When an expert says 95%, is there a range of uncertainty associated with his number? The experts may be from different fields of expertise. They may have a different utility function as to the value of evidence. For example an expert in biophysics may give small credibility to epidemiological evidence because they feel controlled studies are more meaningful than uncontrolled studies. Thus two experts in different disciplines reporting 95% confidence may report it for different assessments of the evidence. Therefore, because of the above concerns, it is difficult to interpret the confidence level. The validity and reliability of the confidence estimates are uncertain.

The methodology attempts to reconcile the confidence of all the experts into a uniform result. They provide a range, particularly for non-agreements. The derivation of this range is never made explicit. Was it some weighted average and what were the assumptions for aggregation? Consistency among the experts is desired. If there weren't, do we need more experts? Three experts in epidemiology may be insufficient to represent the breath of the available scientific literature. Perhaps the confidence profile method of Eddy (Eddy) would be useful to estimate uncertainty.

The authors did not seem to use sensitivity analysis. Sensitivity analysis evaluates the stability of the conclusions of an analysis to assumptions in the analysis. When a conclusion is shown to be invariable to the assumptions, confidence in the validity of the conclusions of the analysis is enhanced. An implicit assumption of decision analysis

is that the values of the probabilities are the correct values. If the probability values varied, would the aggregate analysis hold? Are the decisions sufficiently robust?

CONCLUSIONS

The authors did an outstanding job of gathering evidence and seemed to apply the Delphi method in a comprehensive manner to help the experts derive confidence levels. However, as discussed, the authors need to do a more rigorous Bayesian network analysis that would include uncertainty analysis, sensitivity analysis, which would help more clearly interpret sources for a lack of consistency and coherence.

Perhaps a panel of experts in each discipline could help formulate different components of the decision tree, from evidence in that specific discipline. A complicated assessment requires as many formidable methods as possible to address the problem. This initial analysis can only serve as a beginning. The Delphi method can be further refined and be used for the different components of the network. Perhaps then, can an acceptable summary from a Bayesian analysis be accepted.

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1. We have taken the position that we are not greatly influenced by arguments based on physics and simplified biological models that suggested that residential and occupational levels of EMF can't possibly produce bio effects. We say that theories should be used to predict results that are falsifiable and should not be used to discount evidence. Thus, our prior degree of confidence is not vanishing small. Do you agree? Please comment

Yes, I agree that the argument that bioeffects are unthinkable because of lack of a mechanistic explanation should not prevent the development of additional hypotheses.. However, I do not quite understand what you mean for "simplified biological models". Would you be influenced (and how?) by arguments based on sophisticated and validated biological models suggesting that EMFs do not produce bioeffects? As a philosophical approach, total agreement about uncorrectness of use of theory to discount evidence.

2. Each of the three core reviewers have laid out their initial (prior) degree of confidence that residential or occupational EMFs could produce relative risks of various sizes. These estimates are constrained by what we know about animal bioassays for cancer and by the lack of dramatic change in disease rates after the introduction of electricity and as the use of electricity increased. Reasons are given for these judgements. Do they seem reasonable? How much higher or lower would you're a priori degree of confidence be for any environmental/occupational agent? For EMFs? Why?

Quite frankly, I hardly understand this question. What we know from animal bioassays might be taken into consideration in the process of hazard identification but much less so for risk estimates. Trends in disease rates in the general population are hardly useful for any inference, in the case of agents associated to low relative risks.

3. We are not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects nor were we convinced of a chain of events that led to pathology. Yet we did not let this pull our degreee of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment

Yes, I fully agree. The weight of biological plausibility cannot be approached univocally. Bradford Hill pointed out that it is one of several criteria contributing to causal inference (i.e. hazard identification), among which the only sine qua non is temporal sequence. Bradford Hill himself cautioned against an excessively rigid approach. , given that "what is biologically plausible depends upon the biological knowledge of the day" Biological plausibility implies that the sign of an association matches with knowledge on biological mechanisms of both toxicity and disease. carcinogenesis in man. A specular consideration applies to what is currently biologically implausible (Bruce Ames stated that for most pesticides, carcinogenicity is implausible on the grounds of their biological properties: I would hesitate to use a statement of this sort for public health decisions). As Caterina Botti and Pietro Comba have pointed out that in the process of causal inference, the relative weights given to "biological" features (mechanistic aspects) and to "statistical" aspects (strength of the association, reproducibility of findings) largely depend on the values held by the individual scientists and that these values are more often implicit than explicit.

As Douglas Weed has stated, the question "how does a plausible mechanism differ from a known mechanism?" remains unanswered. The same applies to the specular difference between an implausible mechanism and a mechanism which can be excluded.

4. We viewed the animal pathology literature as largely null, with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment

Yes I agree. It is plenty of examples of proven causal associations in man which could not be reproduced in laboratory animals(or were reproduced with much delay and difficulties). Major examples outside the domaine of cancer are the Toxic Oil Syndrome in Spain and Eosinophilia-Myalgia Syndrome in the US. Admittedly, these are syndromes. As for more specific conditions, it took decades to find out that the hen was the most suitable species to reproduce delayed neurotoxicity by TOCP. In the case of cancer, expectations that the condition must be reproduced in laboratory animals are conditioned by a hypothetical unifying theory of mechanisms of carcinogenesis. When I was younger, laboratory animals were considered to be refractory to the carcinogenicity of arsenic and benzene. This later proved to be uncorrect.

Quite frankly, I cannot see how for some of you "this pattern of evidence" actually increased the degree of confidence somewhat

5. Not all epidemiologists would agree with our position that relative risks between 1 and 2 should be taken seriously unless there is specified evidence for confounding or bias to explain it away. Do you agree? Please comment

I would distinguish between relative risks of that size on the basis of the underlying studies. Indeed, they may result: (a). as a side finding in an exploratory study, (b). from one or a few studies designed in order to test a hypothesis-related to the particular agent or (c). from properly done meta-analyses based on reliable studies. Under (a), I would dismiss the observation (unless I am aware of some mechanistic observations which render it plausible) . Under (b), I would take it seriously. Under (c), I would take it very seriously, up to assert that the association is causal, particularly if there are two or more independent proper meta-analyses based on different sets of reliable studies.

6. We said that a lack of specificity in the association of EMFs with subtypes of cancer and evidence for effects on various types of disease did not pull down our degree of confidence and might even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature. Do you agree? Please comment

Again, I would establish a difference between two different scenarios:

- *are we looking for the whole spectrum of target organs of the carcinogenicity of an agent which is already known to produce cancer in some organs (eg target organs of the carcinogenicity of asbestos other than lung and pleura) or*
- *are we trying to infer about the carcinogenicity of an agent for which all the (statistical) evidence we have derives from the joint consideration of several target organs?.*

In the latter case, which is the case of EMFs, my (old age) instinct tells me to be cautious. In carcinogenesis experiments on animals, there are examples of a wide spectrum of action (N-nitrosomethylurea, just to make an example which is familiar to me). This applies to direct acting carcinogens. For humans, and for carcinogens

requiring metabolic activation the scenario is "unusual" (which does not mean that it does not occur). Admittedly, I am biased towards chemical carcinogens. Ionizing radiation (but not UV) are different. EMF are a question mark.

Again, I would work out my reply on a case by case basis: how many independent studies are available? Do they overlap in the individual target organs contributing to the overall (statistical) evidence? If this is the case, effects on various types of disease should not pull down your degree of confidence. Still, I see no plausible (excuse me for using this term) reason for increasing your degree of confidence compared to the more conventional situation in which you only have an excess in one organ.

Admittedly, I am influenced by my feeling (if I am not wrong) that for all known/or suspected human carcinogens, both the first clue and the first convincing causal inference were organ specific.

7. Have we done an adequate job in presenting the arguments for and against causality or are we assigning weak arguments to the "con" or the "pro" position?

You have done an excellent job, it is obvious that your readers will use their individual degrees of freedom in deciding how weak is any individual argument

8. Our Risk Evaluation Guidelines (REGs) define some "plain language phrases" to express our degree of confidence. However, when we actually applied them we found they were not problem free:

a) Some of the phrases are not mutually exclusive. For esxample, Possible >50% overlaps "highloy problable" and virtually certain. "Possible < 51% overlaps "Possible 50%". In this case, the overalp is slight, but important, since it is about the "balance of probability".

b) These phrases are grammatically awkward and they are not really "user friendly". How could we rephrase them, without violating the spirit of the REGs? Please write any suggestion next to each phrase:

Confidence range	*Current phrase*	*Suggested alternative*
>98%	*Virtually certain*	
90-98%	*Highly probably*	
50-90%	*Possible > 50%*	
10-50%	*Possible < 51%*	
2-10%	*Very improbable*	
<2%	*Virtually certain that it is not causal*	

I have no suggested alternatives. I am as unhappy as you are about your terms. Perhaps times are premature for a systematic approach to subjective probability. Rather than searching for algorithms and definitions, shouldn't the whole issue be discussed with the decision makers in narrative terms? In any case, I am ready to test your approach in some European context.

Dr Raymond Neutra,
Chief
Division of Environmental and Occupational Disease Control
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1515Clay Street, Suite 1701
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Re: Review of the third draft of the Electric and Magnetic Field (EMF) Risk Evaluation and Policy Options document.

Dear Raymond,

Thank you for inviting me to review the third draft of your EMF document.

I find this document very well done, very instructive and very comprehensive. I am most impressed by the meticulous care given to the literature assessment and the major efforts invested to adhere to objectivity in interpreting this literature. It is very revealing to see each reviewer's judgment in the light of the evidence. It gives sound justification to their assessment.

On the negative side, I have little to say.

In general, I found the "Statements to the Public" too cautious. Quite often, it is as if you are apologetic for having come to the conclusion that the review points in the direction of the existence of a risk. I take as example the statement on Suicide on age 280. "Using the guidelines …California EMF Program they concluded that it is "10 to 50% likely" that exposure to EMFs at home or work could add [so far that's fine] slightly [What does this really mean? Does EMF contribute or not to the risk? How do you measure "slightly"? I do not see the value of this qualification.] As this phrase implies, there is also a chance that EMF has no effect on this risk at all. [This sounds apologetic or worse implies some outside pressure on the writers of the document]."

The repetition of the sentence "As this phrase implies, there is also a chance that EMF has no effect on this risk at all." is troubling to the reader of the document. It should be stated once at the beginning of the report but not repeated over and over again.

In general, I concur with the views expressed in this report and I congratulate the reviewer and yourself for this document that should become a classic of the EMF questions.

Here are my answers to the set of questions that you raised in your transmission letter.

1- *About discarding arguments based on physics and simplified biological models that negated EMF bio effects on the basis that they are of limited value in face of evidence*. I surely agree with this view. I congratulate you for having the courage of stating it openly. However, one remarkable feature of the entire research on the health effect of EMF has been the wide multidisciplinary approach taken to study it. Rarely in history has a health question been studied by so many diverse disciplines and by so many scientists of varied background in such a short period of time. This has lead to a very rich body of knowledge and has forced each discipline to go beyond its own limits in addressing the question and in scrutinizing the results. The confrontations of scientists' viewpoints on the same topic could only have lead to a collective enrichment that should be acknowledged in the history of research on EMF and health.

2- *Change in my degree of confidence of a causal relationship between EMF exposure and some health issues.* The reasons underlying the initial (prior) degree of confidence by the three core reviewers are very

reasonable. The three reviewers did not start with the same degree of conviction. At the end of the assessment, the ranking of conviction between the three had remained the same, but each one had seen his conviction gone up by some margin. This testifies to the quality of the assessment done.

As for my own reaction, the reading of the report has increased my degree of conviction that the association between leukemia and EMF is not the result of biases or confounding or chance. In that sense, it has increased my belief in a causal relationship but I am still puzzled by the inconsistencies between studies as much on the exposure side as, and even more, on the disease side. The type of leukemia associated with EMF vary quite a bit between authors and are not necessarily reflected in the rate of overall leukemia. I will come back on this issue latter.

Concerning the adult leukemia studies, I notice that the report lumps together the occupational studies with the residential studies. I think that the comparison with childhood studies [that are essentially residential in nature] should be made with residential adult studies. When one does this, one realizes that the residential adult studies yield results that are very comparable with the childhood studies on leukemia. I have published my views and analysis on these previously. [**Theriault G**, Li CY. Risks of leukaemia among residents close to high voltage transmission electric lines. *Occup Environ Med* 1997;54:625-628.]

On other health outcomes, my a priori degree of conviction has remained quite the same after the reading of the report. Contrary to leukemia, the other health issues have not been studied enough to come to a definitive conclusion. I am impressed by the results of the studies on Amyotrophic Lateral Sclerosis (ALS) and await other original contributions.

3- *Epidemiological evidence vs. lack of mechanism.* I fully concur with the view that the availability of a mechanism of action or a chain of events that leads to pathology would contribute strongly to recognizing a causal relationship between some health outcomes and EMF exposure, but their absence do not negate the evidence as seen by epidemiologists and can only serve as a stimulus to keep searching for such a pathological mechanism.

4- *About the non-contribution of animal studies.* There are other examples where animal studies have contributed little to supporting known cause of diseases in human. The classical example is the inability to reproduce lung cancer among animal exposed to asbestos. A comparable example has been the generation of leukemia by exposing animals to benzene.

5- *About the size of the relative risk.* Why would a study with a risk of 1.0 or even 0.8 been considered perfectly acceptable as showing the absence of a risk and a study with a risk of 1.5 or below been immediately judged as suspected of being flawed by biases and confounding. The size of the risk as nothing to do with the quality of a study. If a true risk is small, a well-done study will yield a small risk. If the study is powerful enough, this small risk will become 95% certain. I like the way you have handled this question in the argumentation under section 8, leukemia. The reply was that with so many studies from so many diverse populations and conducted by so many investigators, the same biases and confounding cannot have taken placed all the time and act in the same direction.

6- *About the lack of specificity*. I am more worry about the lack of specificity. This applies more acutely to leukemia. It is puzzling to realize that some studies observed increase in the risk of one leukemia sub-type and other studies in another sub-type, with or without an increase in the risk for overall leukemia. This lack of specificity pulls down my degree of confidence. I would need some reasonable explanation that I have not found so far. I think that the report does not discuss well the lack of specificity and the reviewers are wrong in using the lack of specificity as “even increase our degree of confidence that epidemiological associations between disease X and EMF are causal in nature”.

7- *The pro and against arguments*. I am in agreement with the arguments proposed. In general, they are well balanced and the "comment and summary" column sheds a neat light on where a reasonable person would stand. But very likely, opponents of the epidemiologists' view will question the value of the positions taken. The document, by its approach and the background of the three reviewers will be seen as the epidemiologists' "Bible" on health effect of EMF.

8- *About the chosen classification and overlapping of the categories*. I am not troubled by your classification. A layperson will understand the language easily. The only change that I would propose is to replace Possible <51% by Possible <50% and Possible >50%, Possible ≥50%.

Page by page comments

Page #	Table #	Line #	Comments
5		21-22	The sentence seems to imply that the mentioned EMF mixture have been seriously studied which is not the case. Make it clearer that the less supportive evidence may be the result of inadequate testing so far
28		36	Antenna a have
86		McDowall 1983	Error in columns AML and ALL
94		Column #1, (A3)	Not a clear statement. Can the point be made easier to read?
96		Column #2, sentence #4	You should not use mechanistic arguments in favor of causality whence you have taken the view of discarding the mechanistic studies overall.
100		6	Reader will be is
108	8.4.9	Line 7	Analyses of child …
207	Box	3	To be Ainadequate@ and

My review is not as thorough as I would have liked. There are some sections that I scrutinized rather carefully and others that I glimpsed through. I hope that my comments will help you in improving this already very remarkable piece of work.

Regards,

Gilles Theriault
Professor,
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1020 Pine Avenue West
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Haven't we had enough of this silliness?

D. A. Thompson
1905 8th Street
Harlan, Iowa 51537

RE: EMF Risk evaluation

To: Dr.Richard Neutra; Mr.Jack Collins

Dear Ray,

I probably misunderstood you, because when I said that I would be pleased to review your document, I thought that you would send me a summary and the evaluations, and not an imposing, bulky document. While I wish to congratulate you and your associates for the tremendous amount of work you did and your very remarkable achievements, I hope you will forgive me for not reviewing and commenting the entire volume page by page. It would take quiet a long time, and you may instead need some comments in a hurry, as in addition I have several other commitments.

I will submit to you some comments, hoping they might be useful to you. I will begin (a) with a tentative brief reply to the 8 points in your letter of July 9,2001, and will follow (b) with a few comments on specific pages of your document.

(a)

1.I agree that arguments based on physics and simplified biological models should not have a prevailing influence on your evaluations. However, the statement concerning falsifiable results sounds as popperian extremism. I would delete it.
2. I agree that the lack of understanding of the possible mechanism of action of EMF should not influence negatively a priori your confidence in the epidemiological findings.
3. I agree that the available animal data are of little significance, and I also agree that this should not pull down the confidence in the epidemiological findings. I would add that the lack of relevance of most of the animal data is due to the fact that most of the tests on EMF were conceived and carried out as if EMF were a chemical or chemical mixture. Different approaches may be, actually must be, explored.
4.I agree in absolute that a RR between 1 and 2 should be taken seriously; in the specific case of EMF the existence of confounding or bias, although not entirely dismissed, does not seem to play a role. The precautionary principle suggests that evidence for a risk associated with an exposure to EMF should be taken very seriously.
5. That various types of cancer and various types of disease appear to be associated with exposure to EMF is plausible, but I would not take it, at least at present, as a reinforcement of the degree of confidence in the available epidemiological findings.
6. I believe you have done a good job in presenting arguments in favor and against, although a certain degree of preconceived conviction may transpire. I would add that this is almost unavoidable, and welcome, for anybody who cares about public health.
7. I am not convinced of the usefulness of quantifying the confidence ranges in the way you propose. Although the IARC categorization in 1, 2A, 2B and 3 is far from perfect, I don't think that you reach much more by the percentages you propose. For instance, I don't see how one may decide when to keep out some of the 50-90% from the higher category? You just add one more category to those of IARC, and use numbers (percentages) instead of words. We have battled for years on those words, but nobody seems to be able to come out with a better classification, yet. The only reason in favor of using your percentages would be if they could actually help regulatory people to come to reasonable decisions. I am unable to evaluate that site of the issue.

(b)

The distinction between the confidence that an effect is causal and the probability that individuals exposed develop the disease is essential, but it seems overemphasized. I don't recollect of having ever been confronted with anybody unable to make such a distinction (paged 9 and others).

The IARC classification is not necessarily driven " by the stream of evidence of animal pathology studies" (page 9). As a matter of fact the assignment to group 1 totally, and to group 2A largely depend on epidemiological results. Even in group 2B human data play a relevant role.

There are some apparent contradictions: on paged 15 childhood and adult leukemia appear to be in the same category, but this is not so on page 13 and in table 1; male and female breast cancer are sometimes placed in the same, and in others in different categories.

I am not sure that the distinction between the criteria adopted by IARC and those of the California program (page 14) is actually helpful, or even correct. The IARC system, based on the quality of evidence, takes into account the degree of confidence as it is clearly explained in the preamble to each Monograph.

The IARC has always emphasized that the evaluations are a matter of scientific judgment, and cannot be translated ipso facto into regulations, as they represent only part of the body of information on which regulatory measures are taken. The California evaluations are by necessity more directly addressed toward regulations. This said, I may share with you the feeling that IARC has been sometimes too shy or exaggeratedly prudent in assessing risks based on evidence from some epidemiological studies or case-reports, with the result of minimizing them. This type of prudence does not encourage the adoption of measures of primary prevention and is not in favor of public health.

With renewed congratulations for the excellent work you have done, and kind regards

Lorenzo Tomatis, M.D.
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For comment file

-----Original Message-----
From: Jim Tucker [SMTP:tucker5@llnl.gov]
Sent: Wednesday, August 08, 2001 9:54 AM
To: rneutra@igc.apc.org
Subject: comments for consideration

Dear Ray,

I'd like to provide a very brief summary of my thoughts and concerns about the conclusions to be communicated in the final EMF report. I am hoping that the report will make it clear that the relative risks of health effects of EMF exposure are very low, and that the confidence intervals around the point estimate should include zero increased risk. I am comfortable with the point estimate being greater than zero risk, but the "grayness" of our uncertainty must be reflected in the language describing the risk. Furthermore, this language must indicate the distinct possibility that there is no increased risk whatsoever.

I remain concerned about some steps in the process used thus far. As I stated yesterday, considerable importance should be placed on understanding underlying mechanisms because the absence of a mechanism makes it harder to establish a relationship between exposure and outcome. To say the same thing a different way, the priors associated with EMF must be very low, say 1-2%, because of the absence of solid theory to support a plausible mechanism. This, coupled with the inconsistent findings provided by the various streams of evidence would result in a similarly low posterior probability accompanied by wide confidence intervals.

I also remain concerned that there are biases in the manner by which various streams of evidence were accorded value. It seems that the epidemiological evidence is weighted much more heavily than the biophysical, physiological, mechanistic, or animal pathology work. The differences in the ways these streams of evidence are valued must either be justified more extensively or adjusted to account for their value more fairly. How is it that "negative" results for some streams of evidence don't reduce the posterior probability, but can only increase it? Are there any laboratory results that would decrease the posterior probability? This question was raised at the meeting yesterday and I'm not sure it got answered.

That's it. The meeting was good and I thought it was productive.

Jim
Jim Tucker, Ph.D.
Lawrence Livermore National Laboratory
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I have only a few comments on the 3rd draft. These are rather global, but I have called out specific places where I believe the problems are evident.

Jim Tucker
Lawrence Livermore National Laboratory

Page #	Line #	Comments
5	48 ff.	The "burden of proof" for biophysical effects should not be considered "unusual" simply because ambient levels of other pollutants often don't produce effects large enough to see in the laboratory. The key difference here is that chemical pollutants have plausible mechanisms of action based on well-accepted knowledge of interactions between cells, DNA, and chemicals. This is in marked contrast to EMF for which our knowledge of biophysics predicts that no effects are possible. Said another way, there would have to be a significant gap in our knowledge of EMFs for a biophysical effect to have a plausible mechanism of action. The question then becomes one of determining whether such a knowledge gap exists. If "truth" is that there is no knowledge gap (i.e., EMF cannot cause biological effects) then the prior degree of confidence must be zero. To the extent that we believe a knowledge gap may exist, the prior degree of confidence should increase.
7	8 ff.	To me it seems quite inappropriate to allow some scientific arguments to be persuasive but not others. The biophysical arguments must be given their full weight and not excluded from consideration. To do otherwise is to instill a bias in the analyses.

Answers to the questions posed in the letter dated July 9, 2001.

By Jim Tucker, Lawrence Livermore National Laboratory

1. I agree that theories should not be used to discount evidence. I also agree that the prior degrees of confidence should not be zero. However, I feel that the prior degrees of confidence should be quite small, say 1-2%. This is based on my belief that our understanding of biophysical principles is quite good and that there is not likely to be a gap in our knowledge of biophysics that would leave room for EMF to cause biological effects.

2. As noted in my answer to the first question, my prior degrees of confidence would be lower than each of the three reviewers, i.e. in the range of 1-2%.

3. No, I do not agree that mechanistic effects of EMF should not reduce confidence in biological outcomes. To me it seems quite inappropriate to allow some scientific arguments to be persuasive but not others. The biophysical arguments must be given their full weight and not excluded from consideration. To do otherwise is to instill a bias in the analyses.

4. If "null" means that the body of evidence is ignored, then I agree, but only if the experiments were done poorly, in which case there is a scientific basis for their exclusion. However, if "null" means that the body of evidence is considered scientifically valid but that the results show no biological effect attributable to EMF, then these data need to be included in the analyses, just as do studies that claim an EMF effect.

5. I would tend to agree that small relative risks should be taken somewhat seriously, providing that the confidence intervals are small enough to exclude 1. However, small relative risks should not be taken as seriously as larger risks. The extent to which relative risks are taken seriously should scale with the size of those risks. Similarly, the extent to which Society expends its public resources to solve a problem should scale with the magnitude of that problem. More resources should be used to address a relative risk of 2 than a relative risk of 1.2.

6. The lack of specificity between EMFs and subtypes of cancer could be argued as either reducing or increasing the degree of confidence of associations. In this case I'm not sure which is more appropriate, so I would tend to agree with the approach that was taken.

7. I believe you have done an adequate job of presenting the arguments for and against causality.

8. For this Table, I would be inclined to keep the words in column 2 as they are current written. However, I suggest that the number ranges in column 1 be changed slightly to avoid overlap, as follows:

Confidence range	New confidence range
>98%	(same)
90-98%	90-98%
50-90%	50-<90%
10-50%	10-<50%
2-10%	2-<10%
<2%	(same)

CAUSE
California Alliance for Utility Safety and Education
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San Diego, California 92130

September 10, 2001
Raymond R. Neutra, M.D., Dr.P.H. Chief
Division of Environmental and Occupational Disease Control
Department of Health Services
1515 Clay Street, Suite 1701
Oakland CA 94612

Comments on the Risk Evaluation and Policy Options in the Face of Possible Risk from Power Frequency Electric and Magnetic Fields Draft Document of April 2001

Dear Dr. Neutra:

The following is CAUSE's response to the above reverenced documents. We were unable to submit our comments in the form requested by you in your letter of July 9, 2001 because the word "involuntary" appears only one time in the entire document.

Involuntary Exposure.

1. Given the uncertainties about EMFs, informed individuals are ethically free to make their own choices about whether or not to limit their own exposures. But what policy options relate to issues where parents, patients, children, teachers and employees are involuntarily exposed or information is withheld from these populations about their involuntary exposure to EMF?
2. One of the policy recommendations of The Report of the California EMF Consensus Group states that "people who are concerned about EMF consider exercising reasonable judgment in educating themselves on issues of EMF and deciding if they wish to avoid EMF exposure. "….People may elect to avoid unnecessary EMF exposure according to their individual values, beliefs, and resources. " The Policy Option should at the very least provide detailed guidance on how to do this.
3. It would be useful to have the report compare the involuntary EMF exposure to other involuntary exposures of other known hazards.
4. The report does not address the "no regrets" policy which is designed to balance scientific uncertainty, level of public concern and preparation for an uncertain future. This policy allows for taking action now that appropriately uses scarce public resources so that decisions made today will be seen as appropriate under a wide range of future scenarios. The level of public concern is especially high when there is a combination of uncertainty and involuntary exposures because there is no choice in the acceptance of the risk. The Policy Options should address this "riskier" perception and include an analysis what actions could be recommended to alleviate the added anxiety due to the involuntary nature of some EMF exposures.

Sincerely,

Joan I. Tukey

Mr. Collins:
The report on Electricity that your organization has come out with breaks ground for the future of personal health to The United States human population both now and in the future!!

We know too well the importance of what you have done as our dairy farm suffers with stray voltage!!

Keep up the work with power quality, you are doing the "RIGHT" thing.

Brice/Sharon Wagner Elk River Dairy, Port Orford, Oregon established in 1915!!

Daniel Wartenberg
19 Stouts Road
Skillman, NJ 08558
September 10, 2001

Comments on, "An Evaluation of the Possible Risks From EMFs From Power Lines, Internal Wiring, Electrical Occupations and Appliances

Due to time constraints, my comments are limited to the approach used and one specific disease outcome, childhood leukemia. If more time were made available, I would consider reviewing additional sections of this complex but interesting document.

The Approach (Chapter 1)

The literature on the possible association between exposure to EMFs and adverse health effects was considered by three California Department of Health Services staff members. They used both the California Guidelines, which allow consideration of emerging evidence require an assessment of quantitative degree of confidence, and the IARC guidelines, which are based on a quality of evidence evaluation.

The approach used to conduct an evaluation rather than yet another review of the literature is admirable if a bit confusing. Review after review have provided fairly similar results but required substantial resources of time and effort. This report tries to go beyond the previous reviews and develop more useful information for policy-makers. The limitation of this approach is that it may be difficulty to follow for those not familiar with the details of the literature on this topic.

Another problem with this approach is difficulty in communicating the complexity of the information collected. The amount of effort and degree of detail afforded by this approach is impressive and surpasses that of previous reviews. The liabilities are that opinions and expert judgments offered were developed by only 3 scientists, as compared with the other reviews that had far more participants, and that the complexity of the information is probably overwhelming except to those most actively in EMF research. I wonder if the richness of information provided makes decision-making easier or more difficulty for policy-makers who may not be as well schooled in the nuances of experimental and observational science.

The consideration of prior beliefs of the investigators is an interesting approach to the evaluation of scientific data. It adds a second dimension to the consideration of the final opinion of the reviewer. That is, in addition to the reviewers' final assessment one can evaluate how much individual's opinion changed as a result of considering the newly reviewed studies. Interpreting that, however, is somewhat problematic. If someone was very skeptical and now is on the fence, it that more compelling that someone who was on the fence but now is fairly confident of a causal relation? How does one compare the change versus the actual position of the opinion? While Bayes offers one approach, others are plausible.

Providing information about how reviewers formed their opinions is interesting, academically. It offers much rich information for the study of opinion formation in general and EMF in particular. However, the novelty and complexity of this process will make it difficult for decision-makers to use. It is easier to model policy based on past decisions that it is to adopt a new approach on a highly controversial issue with somewhat inconsistent data and very strong differences of opinion.

Epidemiology of the Leukemias (Chapter 8)

Tables 8.2.1-8.2.14 that list for many of the unresolved issues about EMFs the many arguments against causality, for causality and then comment and summary are excellent. It captures much of the conversations that go on among scientists, organizes them and brings the information together in an extremely useful format.

Through these tables, most of the key issues are addressed in a pro/con format with the conclusions of the reviewers. That, together with the statements of each reviewer, provides useful insight into their final determinations. Again, I am less clear about the utility of the final numerical estimates of certainty.

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September 10, 2001

Via Email (JCollins@dhs.ca.gov)

Mr. Jack Collins
Program Administrator
California EMF Program

Re: Comments on "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances," Draft 3 for Public Comment, April 2001.

Dear Mr. Collins,

On behalf of the Utility Health Sciences Group (U.H.S.G.), we have enclosed the comments of Dr. Mark Israel, Dr. John Boice and Dr. Robert Tarone on the California EMF Program's Draft Report "An Evaluation of the Possible Risks from Electric and Magnetic Fields (EMFs) from Power Lines, Internal Wiring, Electrical Occupations and Appliances," Draft 3 for Public Comment, April 2001.

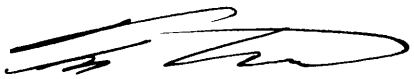
These comments are submitted to CDHS, the California EMF Program and your attention pursuant to the public comment provisions identified in the draft CDHS EMF Report.

The Utility Health Sciences Group is a group of electric utilities interested in promoting open dialogue on health issues including electric and magnetic fields.

Dr. Israel, Dr. Boice and Dr. Tarone were asked by U.H.S.G. to conduct independent professional reviews of the draft CDHS EMF Report and to prepare comments based on their reviews.

Thank you for your consideration of these comments.

Very Truly Yours,



Tom Watson

Professional Experience of Commenter:

Mark A. Israel, M.D.

Director, Norris-Cotton Comprehensive Cancer Center, Dartmouth Medical School, Hanover, New Hampshire, September 2001 - present.

Professor of Pediatrics and Genetics, Dartmouth Medical School, September 2001 – present.

Director, Preuss Laboratory of Molecular Neuro-Oncology, University of California at San Francisco (UCSF), 1990 – 2001.

Kathleen M. Plant Distinguished Professor, University of California, San Francisco, 1997 – 2001; Professor of Pediatrics and Neurological Surgery, UCSF Medical School, 1990 – 2001.

National Cancer Institute Board of Scientific Counselors, 1997-2001.

Head, Molecular Genetics Section, Pediatric Branch, National Cancer Institute, National Institute of Health, 1984 – 1989.

Scientific Advisory Board, The Children's Cancer Foundation.

Medical Advisory Board, Pediatric Brain Tumor Foundation of America.

Scientific Advisory Council, American Brain Tumor Association.

External Scientific Advisory Board, Wisconsin Comprehensive Cancer Center, 1995-1998.

Board of Trustees, Leukemia Society of America, Inc. Washington, D.C. Chapter, 1985-1989;

Board of Directors, Federation for the Advancement of Education in the Sciences, 1984-1989.

Farber Award, 1998.

Heinz Karger Memorial Foundation Prize, 1988.

U.S. Public Health Service Commendation Medal, 1985 & 1987.

Associate Editor, Cancer Research, Clinical Cancer Research, Journal of Pediatric Hematology and Oncology, Molecular and Cellular Differentiation, Oncology Reports, The Journal of Experimental Therapeutics and Oncology; Editorial Board, Neuro-Oncology.

200+ publications on the molecular genetics of cancer, cancer causation and pediatric cancers.

M.D., Albert Einstein College of Medicine, 1973. Board certified in Pediatrics and Pediatric Hematology-Oncology.

Mark Israel, M.D.

Comments

on

Draft California EMF Program Report

"An Evaluation Of The Possible Risks From Electric And Magnetic Fields (EMFs) From Power Lines, Internal Wiring, Electrical Occupations And Appliances"

I am a pediatric oncologist, Director of the Norris Cotton Cancer Center at Dartmouth Medical School, and Professor of Pediatrics and Genetics at Dartmouth Medical School. From 1990 until this August, I was Director of the Preuss Laboratory of Molecular Neuro-Oncology at the University of California at San Francisco (UCSF) and Professor of Pediatrics and Neurological Surgery at UCSF Medical School. I have conducted an independent review of the draft California EMF Program Report and respond below to certain questions raised by CDHS about the draft Report.*

CDHS Question No. 1:

> "We have taken the position that we are not greatly influenced by arguments based on physics and simplified biological models that suggested that residential and occupational levels of EMFs can't possibly produce bio effects. We say that theories should be used to predict results that are falsifiable and should not be used to discount evidence. Thus, our prior degree of confidence is not vanishing small. Do you agree? Please comment."

Comment:

I do not agree. The large body of experimental data seeking evidence of a relationship between EMF and biological effects related to cancer causation is widely interpreted as failing to identify such a relationship. In addition, significant questions have been raised as to whether power frequency EMF at environmental levels are capable of imparting sufficient energy to have adverse effects on living organisms. Thus, the appropriate hypothesis based on experimental evidence is that power frequency EMF do <u>not</u> cause biological effects related to cancer, and the prior degree of confidence should be "vanishing" small. While evidence to the contrary should be considered, any such evidence must be interpreted in light of existing physical theory and the scientific evidence on which such theories are based.

The decision not to be influenced by arguments based on physics or "simplified biological models" ignores that both of these approaches are well-established and are fundamental scientific tools for seeking evidence of causal relationships. In testing hypotheses, good science must take into account existing scientific knowledge. When a

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* These comments are not offered on behalf of any institution with which I am associated.

hypothesis (such as the hypothesis that power frequency EMF cause biological changes related to cancer) appears to be in conflict with the principles of physics, extremely strong and consistently reproducible experimental evidence is required to validate the hypothesis. Given that the experimental evidence in this instance is neither strong nor consistent, it is particularly inappropriate to simply bypass the question of physical plausibility in evaluating the hypothesis. Similarly, the use of model systems is a key means of eliminating confounding variables in evaluating potential causal relationships. Virtually all aspects of disease causation are routinely studied in model systems. For these reasons, an evaluation of EMF and cancer causation should include an integrated analysis of experimentation using model systems and the questions raised by application of the principles of physics. The lack of such an analysis is a significant omission in the draft CDHS Report.

CDHS Question No. 3:

> "We were not deeply convinced of mechanistic explanations of how EMFs could cause bioeffects nor were we convinced of a chain of events that led to pathology. Yet we did not let this pull our degree of confidence in the epidemiology down much on the grounds that lack of mechanistic understanding is not sensitive or specific. Do you agree? Please comment."

Comment:

After hundreds of studies over many years, the *in vitro* laboratory data fail to demonstrate that EMF is involved in the initiation, promotion or progression of cancer of any kind. Most importantly, despite extensive experimentation, the *in vitro* research has not shown that power frequency EMF is capable of causing damage to the genetic material of the cell (the DNA and chromosomes) that is known to be necessary to produce cancer. While the draft CDHS Report correctly notes that "there is no consistent pattern supporting genotoxicity" and "there is overwhelming negative evidence against DNA damage and chromosomal effects," these statements are essentially cast aside by the subsequent conclusion that "overall the picture is mixed and *does not affect our confidence level much*." The draft CDHS Report's complete discounting of the importance of the *in vitro* research is unwarranted.

Strikingly, the draft CDHS Report's chapter on *in vitro* research is only 2 pages long. The brief discussion of research in this chapter does not identify even a single study relevant to the molecular biology of cancer. By failing to address this important body of research in any meaningful way, the draft CDHS Report leaves the impression that either 1) there is no significant body of *in vitro* research, which is at best misleading, or 2) the *in vitro* research is not important in the overall evaluation of cancer causation, which is scientifically unjustifiable.

The draft CDHS Report's discounting of the *in vitro* research is contrary to the approach taken by the National Institute of Environmental Health Sciences (NIEHS) in directing the recently completed six-year, $46 million national EMF research program (EMF-RAPID). As noted in the 1999 NIEHS Director's Report to Congress, a "major focus" of the EMF RAPID Program "was research that targeted examination of *in vitro* effects that

might clarify potential mechanistic actions of ELF-EMF in order to explain reported epidemiological associations with magnetic fields." NIEHS emphasizes that the EMF-RAPID program focused on this type of controlled laboratory research because this research is an important element in the evaluation of cancer causality. Thus, as the NIEHS Director's Report made clear:

> [t]he NIEHS health effects research program focused on mechanistic, cellular and laboratory studies in the areas of Neurophysiology, behavior, reproduction, development, cellular research, genetic research, cancer and melatonin. ... Mechanistic, cellular and laboratory studies are part of the overall criteria used to determine causality in interpreting epidemiological studies. (emphasis added)

In reviewing the large body of *in vitro* research, the NIEHS Director's Report concludes that "most of the mechanistic work done in cells fail to support a causal relationship between exposure to ELF-EMF at environmental levels and changes in biological function or disease status." NIEHS considers this lack of evidence in the *in vitro* research a factor which "severely complicates" the interpretation of the epidemiologic research.[1]

The National Academy of Sciences (NAS) has also reviewed the results of the EMF-RAPID program. A 1999 report from the NAS National Research Council noted:

> When the EMF-RAPID program began, emphasis was placed on two important phenomena – cancer promotion and gene-related effects *in vitro.* Experiments supported by EMF-RAPID provided some evidence to support, and considerable evidence to refute the view that power-frequency MFs can have biologic effects. Evidence of any robust and replicated effects on the development of cancer is lacking. (emphasis added)

Rather than ignoring the *in vitro* research, the NAS Report concludes that the failure to demonstrate that EMF cause biological effects related to cancer causation is evidence against cancer causation. Thus, NAS concludes that "in view of the negative outcomes of EMF-RAPID replication studies, it now appears even less likely that EMFs in the normal domestic or occupational environment produce important health effects, including cancer."

In my experience as a cancer researcher and as a past member of the Scientific Advisory Board of the National Cancer Institute, the approach taken by the NIEHS and NAS to include analysis of *in vitro* research is standard practice. For the authors of the draft CDHS Report to treat this research as lightly as they have is a significant shortcoming and compromises the opportunity for CDHS to provide a complete and accurate assessment of the body of EMF research relevant to cancer.

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[1] An important omission in the draft CDHS Report is a rationale for not mentioning or relying on the findings of the 1999 NIEHS Director's Report on EMF, given the draft CDHS Report's citation to the 1998 NIEHS Working Group Report, which was a small part of the overall evaluation of EMF research conducted by NIEHS.

CDHS Question No. 4:

> "We viewed the animal pathology literature as largely null, with the exception of the breast cancer promotion studies of the Soviets and Loescher's group and the various experiments with chick embryos. Once again because of arguments given we did not let this pattern of evidence pull down our degree of confidence in the epidemiological literature much and for some of us it actually increased the degree of confidence somewhat. Do you agree? Please comment."

Comment:

The draft Report's chapter on animals studies concludes that "[o]verall, the animal pathology studies are predominately, but not entirely, negative." As noted in Question 4 above, the conclusion that the animal research essentially provides no support for cancer causation, however, is essentially ignored or, more surprisingly, is used to increase rather than decrease the degree of confidence in the suggestive data from the epidemiologic studies. This dismissive approach to the animal data is entirely unjustified.

Animal studies play an important role in the assessment of carcinogenic potential. These studies are used routinely in cancer research to assess the causative potential of many different sorts of agents and are a principal scientific method for determining carcinogenicity. The results of these controlled laboratory experiments should be given significant attention in any evaluation of cancer causality.

The 1999 NIEHS Director's Report on EMF noted the importance of the animal research on EMF and carcinogenicity.

> Animal carcinogenicity studies are routinely used to identify environmental agents that may increase cancer risk in humans. Many areas of biological investigation are more efficiently studied in animal models than in human beings. ... The laboratory data in animal models are inadequate to conclude that exposure to ELF-EMF alters the rate or pattern of cancer. ... [I]t is noteworthy that these data provide no support for the reported epidemiological findings (discussed earlier) of increased risk for leukemia from ELF-EMF exposure. (emphasis added)

Similarly, the 1999 NAS Report on the EMF-RAPID Program concluded that:

> The EMF-RAPID biologic research contributed little evidence to support the hypothesis that a link exists between MF and cancer. The results of the *in vivo* studies do not support an MF effect on cancer initiation, promotion, or progression, and they should be recognized as important studies in the overall evaluation of potential carcinogenic effects of MFs. (emphasis added)

There is an extensive body of animal research that fails to demonstrate a detectable effect or role for EMF in the initiation, promotion, or progression of tumors. For this extensive body of research to be marginalized in the draft CDHS Report is inconsistent with meaningful scientific analysis. Perhaps more importantly, the failure of the draft CDHS Report to give this important data any meaningful weight in the overall causality analysis presents a misleading view of the research for the general public and regulators who may rely on the CDHS Report for information about EMF.

CDHS Question No. 8:

> "Our Risk Evaluation Guidelines (REGs) define some "plain language phrases" to express our degrees of confidence. However, when we actually applied them we found they were not problem free:
>
> a) Some of these phrases are not mutually exclusive. For example, Possible >50% overlaps "highly probable" and virtually certain." "Possible <51%" overlaps "Possible >50%". In this case, the overlap is slight, but important, since it is about the "balance of probability".
>
> b) These phrases are grammatically awkward and they are not really "user friendly". How could we rephrase them, without violating the spirit of the REGs? Please write any suggestions next to each phrase:

Confidence Range	Current Phrase	Suggested alternative
< 98%	Virtually certain	
90-98%	Highly Probably	
50-90%	Possible >50%	
10-50%	Possible <51%	
2-10%	Very Improbable	
< 2%	Virtually Certain	
	that it is not causal"	

Comment:

For the CDHS degree of confidence evaluation to be useful for scientists and the public, it needs to reflect an objective analysis of the weight of scientific evidence. To do so, the evaluation needs to be based on a clearly articulated set of scientific criteria for weighing the underlying evidence. This, however, is strikingly absent. The draft CDHS Report does not provide a description of any such criteria and, to the extent that any criteria were used, the draft Report does not explain how those criteria were applied to the scientific evidence.

This failure to base the degree of confidence analysis on objective scientific criteria is a fundamental flaw in the draft CDHS Report. The use of so-called "plain language phrases" as labels for the various levels of "confidence" does not and cannot remedy this flaw. Without objective criteria for scientists and the public to evaluate, the degree

of confidence analysis in the draft CDHS Report is not a scientific characterization of the relevant research.

Professional Experience of Commenters:

Dr. John Boice

Scientific Director, International Epidemiology Institute, LTD, Rockville, Maryland.

Previous positions include;

Chief, Radiation Epidemiology Branch, Epidemiology and Biostatistics Program, Division of Cancer Etiology, National Cancer Institute, 1984-1986.
Epidemiologist, Epidemiologic Studies Branch. Bureau of Radiological Health, Food and Drug Administration (FDA), DHEW, Rockville, Maryland.
Physicist, Field Studies Branch, Bureau of Radiological Health, FDA, Northeastern Radiological Health Laboratory, Woburn, Massachusetts.

Harvard University School of Public Health, Visiting Lecturer on Epidemiology.

National Council on Radiation Protection and Measurements.

International Commission on Radiation Protection, Main Commission.

Editorial Board, Journal of the National Cancer Institute.

Steering Committee, Childhood Cancer Survivor Study, University of Minnesota.

Advisory Committee for the Harvard Center for Radiation and Health.

Consultant, National Academy of Sciences Committee on the Health Risks from Exposure to Low Levels of Ionizing Radiation.

B.S. Physics and Mathematics, University of Texas at El Paso; M.S. Nuclear Engineering and Science, Rensselaer Polytechnic Institute; S.M. Environmental Health Science (Medical Radiological Physics), Harvard University School of Public Health; Sc.D. Epidemiology, Harvard University School of Public Health.

Dr. Robert Tarone

Chief, Statistical Research and Applications Section, Division of Cancer Epidemiology and Genetics, National Cancer Institute, National Institutes of Health, Bethesda, Maryland.

Statistical Editor, Journal of the National Cancer Institute.

National Institutes of Health Merit Award, 2001.

Division of Cancer Epidemiology and Genetics Exemplary Service Award, 1999.

Epidemiology Review of "An Evaluation of the Possible Risks from Electric and Magnetic Field (EMFs) From Power Lines, Internal Wiring, Electrical Occupations and Appliances," California EMF Program, Draft 3 for Public Comment (April, 2001)

John D. Boice, Jr., Sc.D.

Robert E. Tarone, Ph.D.

September 7, 2001

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Chief, Statistical Research and Applications Section
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* (comments reflect views of author and are not being offered on behalf of any entity with which he is associated)

Epidemiology Review of California April 2001 EMF Report

I. Summary of Review

The California EMF report (April 2001 draft) was reviewed from an epidemiologic perspective as to the evidence for a causal association between low frequency electromagnetic fields (EMF) and cancer, neurological disorders, and miscarriages. Because a new approach called the "degree of confidence in causality" was introduced, we evaluated its underpinnings and potential value. Finally, we pointed out systematic differences between the California reviewers and recent international review panels that evaluated essentially the same, or more recent, data. Additional comments and details are provided in 5 appendices.

In general, we found the "degree of confidence in causality" approach to be too subjective to provide much guidance on whether EMF was causally associated with various outcomes. We also noted that three recent major review panels, including the IARC monograph committee, are much less convinced that EMF is a possible cause of adverse effects in humans than are all three California reviewers. It is difficult to understand how all three California reviewers arrived at positions that are so out of line with those taken by these international review panels.

In brief summary:

1. Degree of Confidence in Causality. The "degree of confidence in causality" approach is too subjective to be of quantitative value. Because of the reliance on the concept of "degree of confidence" throughout the California EMF report, a detailed critique is provided below. One important point to be made is that this approach has no foundation in mathematical or probability theory. The "degree of confidence" is simply a highly subjective device that allows the three reviewers - in the guise of a scientific measure - to attach numbers to their opinions as to the possibility that EMF produces harm. Different reviewers would (and have) come up with different judgments regarding the causal nature of EMF exposures. Further, the "degrees of confidence in causality" presented are not strictly independent, as evidenced by the extent of agreement of the three reviewers. This is not surprising since the three reviewers have worked together for years and likely have similar perspectives.

   Because the "degree of confidence" method is an entirely subjective attempt to quantify risk, it cannot be objectively critiqued. As such, we assessed the conclusions of the California EMF report by contrasting the degrees of confidence of the California reviewers with conclusions reached by recent independent scientific review panels.

2. Recent Review Groups. Recent review panels (IARC, NRPB, NIEHS and Netherlands Health Council) all place substantial importance on the cellular and animal data, which provide little evidence for adverse health effects following EMF exposure. Further, they are influenced by the lack of a plausible biological mechanism by which such low energy fields from residential EMF exposure could cause harm.

3. Cancer. The IARC, NRPB, and Netherlands Health Council have recently reviewed the EMF literature on cancer. Their conclusions are essentially the same, that there is insufficient evidence to conclude that EMF causes adult cancer at any exposure level or childhood cancers at exposure levels less than 4 mG (0.4 microTesla). The only possible exception is for childhood leukemia at exposures greater than 4 mG based on the statistical association seen in recent pooled analyses. Even for childhood leukemia, these review panels suggest that selection bias could have produced the association, and thus were cautious in their classification, i.e., IARC Group 2B, or possibly carcinogenic. The California reviewers are out of line with the conclusions of these mainstream committees for childhood leukemia, adult leukemia, and adult brain cancer. This was especially noticeable for adult brain cancer, where the degrees of confidence of the California reviewers were almost as high as they were for childhood leukemia, yet the epidemiologic evidence for brain cancer provides little support for an association with EMF exposure.

4. Neurological Disorders and Suicide. As reflected in the previous NIEHS and other earlier reviews of neurological disorders and suicide, there is little evidence for an association with EMF, and this is generally reflected in the California review --- with one major discrepancy being for Amyotrophic Lateral Sclerosis (ALS). Reviewing essentially the same literature, the California reviewers all conclude with a high degree of confidence that EMF is a possible cause of ALS. In contrast, the NIEHS Working Group concluded that there is insufficient evidence that magnetic fields cause ALS (24 reviewers agreeing, 1 abstaining, 4 absent). It is unclear why there should be a discrepancy between these reviews, but again this reflects a tendency for the conclusions of all three California reviewers to be out of line with those of independent review groups.

5. Miscarriage. Similar to ALS, the California EMF report judges with a high degree of confidence that EMF is a possible cause of miscarriage, whereas the NIEHS report judges the evidence to be inadequate (for environmental EMF exposure, 22 reviewers voted for "inadequate" and 1 voted "lack of evidence"). The California reviewers, however, apparently relied heavily on two unpublished studies that they were directly or indirectly involved with. These studies report associations between maximum daily EMF exposure level and miscarriage, but as admitted in the California report itself, they are "hypothesis generating" studies that need to be confirmed by others. The two new studies should be interpreted cautiously when assessing the evidence for a causal relationship between magnetic field exposure and miscarriage, because they relied on a novel EMF exposure metric, had very low participation rates, and showed no evidence of a dose-response relationship.

In summary, the "degree of confidence in causality" approach is too subjective to provide guidance as to whether EMF exposures cause human disease. This is evident by the sometimes wide discrepancies between the "degrees of confidence in causality" reported in the California report and conclusions about the strength of evidence for associations between EMF exposure and disease risk recently reached by independent expert review groups.

II. Comments on the *Degree of Confidence in Causality* reported in the California EMF Program evaluation of possible disease risks from EMF exposure

The evaluation of disease risk by the California EMF Program represents the opinions of three scientists from the California Department of Health Services regarding the possible health risks arising from exposure to low-frequency electromagnetic fields (EMF). Apparently, the California Risk Evaluation Guidelines require a quantitative assessment of the level of risk for each exposure and disease under consideration, and to meet this requirement, each of the three scientists provided a *degree of confidence in causality* due to EMF exposure for each disease investigated. The *degree of confidence* has no foundation in mathematical or probability theory (i.e., there is no specified probability space with a corresponding probability measure defined over that space), but nonetheless it seems to be interpreted as a probability in the California report. The *degree of confidence* is, however, simply a device that allows the three scientists to present their opinions as if they are quantitative scientific measures.

The report tries to make an analogy between the *degree of confidence in causality* and a Bayesian posterior probability (pages 21-22), but it is a poor analogy. In a Bayesian analysis one specifies a prior probability distribution on the parameter of interest (e.g., the odds ratio or relative risk of a disease associated with an exposure); this prior probability distribution is always somewhat subjective, but it satisfies all of the requirements for a probability distribution (in particular, the prior integrates to 1.0 over the probability space of the parameter). A model is specified to describe the relationship between the parameter of interest and relevant data that has been collected (e.g., the data from epidemiologic studies of the exposure and the disease of interest), and a likelihood function is calculated based on this model. The Bayesian posterior probability distribution is **calculated** by integrating the product of the prior distribution and the likelihood function. Given the same prior distribution, the same model, and the same data, different people will all arrive at exactly the same posterior probability distribution and posterior probability calculations using Bayesian theory. Different prior distributions will result in different posterior distributions for the same model and data, and sensitivity analyses can be done to indicate the extent to which the posterior distribution depends on the assumed prior.

The prior *degree of confidence* reported in the California EMF Program evaluation is never clearly defined, but is intended to reflect the scientist's prior belief that exposure to electromagnetic fields is capable of causing human disease. The prior *degree of confidence* is not a probability distribution, but is reported as a median (median of what is not clear since there is no specified probability distribution or sample of observed degrees of confidence from which to calculate a median) along with a specified “range” (which is not defined in the report, but presumably represents a range containing all degrees of confidence believed to be plausible by the scientist). The prior *degree of confidence* plays no direct quantitative role in the process of arriving at the final *degree of confidence in causality* for any specific disease, but simply serves as an indication of how likely each scientist was at the outset of deliberations to believe that EMF exposure could cause human disease.

The posterior *degree of confidence* is also reported as a median along with a range. While Bayesian posterior probabilities are **calculated**, the posterior *degree of confidence* is **elicited** (based on training in probability elicitation received by each scientist). As indicated on page 24, the posterior *degree of confidence* is "elicited directly, after a structured consideration of the EMF-specific evidence." The structured consideration of the results of relevant studies consisted of "considering in a narrative fashion the overall direction in which the results tended to push our degree of confidence and the strength of these results..." (page 22). The highly subjective nature of this process is admitted on page 22 of the report, where the end result is described as "our qualitative and semi-quantitative degree of confidence". The posterior *degree of confidence* is somehow elicited by each scientist through a subjective process that includes various considerations and deliberations of experimental and epidemiologic evidence in a narrative fashion. This process also incorporates various personal scientific beliefs (vaguely described in the description of how prior degrees of confidence were arrived at on pages 29 and 30). Thus, the posterior degrees of confidence represent nothing more than subjective attempts to quantify the scientist's opinion. It is important to note that the subjectivity in Bayesian analyses comes only from the specification of the prior distribution, while in the EMF report both the prior and posterior *degrees of confidence* are highly subjective in nature. Sensitivity analyses in the determination of posterior *degrees of confidence* are thus not possible. One can not alter the personal beliefs of each scientist, nor can one wipe out all memory of the considerations and deliberations leading to each scientist's elicitation of a posterior *degree of confidence*. Hence, neither the influence of the scientist's prior beliefs nor the reproducibility of the posterior *degree of confidence* can be evaluated.

The discussions of the process of eliciting priors (pages 29-30) and of the modification of priors based on experimental evidence (pages 14-15) are somewhat instructive, particularly with regard to Reviewer 1. The discussion on pages 29-30 reports prior *degrees of confidence* somewhat different from those presented in Table 1, a discrepancy that is not explained in the report. The prior *degrees of confidence* for disease causation reported on pages 29-30 are 15%, 5%, and 17% for Reviewers 1, 2, and 3, respectively. These compare to the median priors of 12%, 6%, and 10%, respectively, reported in Table 1. The prior of Reviewer 1 is based primarily on the belief that extraneous environmental agents (agents that are not present naturally in nature, but result from human endeavors) are likely to be harmful, and that magnetic fields may affect electrical signals necessary to normal biological functions. The prior of Reviewer 1 would have been higher except for physical arguments showing that the energy levels associated with environmental exposures are too small to cause a biological effect. In spite of the fact that the substantial body of evidence from laboratory and animal experiments is largely negative, Reviewer 1 was convinced by this evidence that biological effects can result from exposure at environmental levels. This conclusion doubled the original prior of Reviewer 1 from 12% (the prior reported on page 15 agrees with Table 1 and not page 29) to 24%. A number of reviews have concluded that there is no convincing evidence of biologic effects of magnetic fields at low levels, and both Reviewers 2 and 3 essentially ignore the experimental results in arriving at their posterior *degrees of confidence*. Thus, the doubling of the *degree of confidence* for Reviewer 1 is difficult to reconcile with the scientific consensus. The penultimate sentence in the section discussing animal studies (page 64) may provide some insight into the reasoning process that led to this doubling. Explaining how the body of animal studies increases slightly the belief that magnetic fields might be harmful, the

sentence states that "a pattern of many false-negative results when the effect is real is more likely than a pattern of a few false-positive studies when the null hypothesis is true." This peculiar statement suggests a lack of understanding of hypothesis testing. Given the large number of studies reviewed, a few false positive studies are, in fact, expected (i.e., the likely result). Even accepting the reviewer's premise that there were many studies of "real effects", unless every such study resulted in a false-negative finding, the number of positive studies should exceed the expected number of false-positive studies, and no evidence is provided that this is the case.

The general agreement of the posterior degrees of confidence of the three scientists (i.e., all three are usually either above 50% or below 50%) is not surprising, given that the deliberations were not done independently. The three scientists are colleagues who work together and write research papers together. They presented their preliminary assessment to the same review panel consisting of other scientists from the Department of Health Services, and revised their degrees of confidence according to the suggestions of that panel. Because they went through much of the elicitation process together, some convergence of posterior degrees of confidence would seem likely. Consistency of opinions of three scientists at different institutions, going through the elicitation process independently, would be more convincing than the general agreement observed among the California reviewers.

Summary: The "degree of confidence in causality" is a subjective device that has no mathematical underpinnings or clear interpretation. It reflects only the opinion of the reviewer and is not a probability or a quantitative scientific measure. The three reviewers place little weight on the lack of cellular and animal evidence for adverse effects from EMF and on the absence of a plausible biological mechanisms. Other groups of scientists, including recent international review groups, consider these factors to be much more important and are much "less confident" that EMF is a human carcinogen. There is nothing wrong in expressing ones beliefs, but it is misleading to present them as a quantitative scientific measure. Further, it cannot be said that the opinions of the reviewers are independent, because the reviewers have worked together for years and went through much of the probability elicitation process together in the attempt to quantify their opinions.

III. Cancer

The "degree of confidence in causality" presented in this report is a new concept that has not been applied previously to the assessment of risk associated with magnetic fields (or any other exposure). Because the degree of confidence in causality is entirely subjective, it is difficult to provide an objective critique of the California EMF Program report. An indirect assessment of the extent to which the reported degrees of confidence are consistent with the deliberations of independent groups of scientific experts is possible, however, by comparing the California EMF Program report to recent summary evaluations of the possible health risks associated with electromagnetic field exposure. There are three recent evaluations of possible cancer risk and magnetic field exposures, all of which considered published papers at least as current as those examined by the California reviewers: an advisory group report to the National Radiological Protection Board of the United Kingdom (2001); an advisory committee report to the Health Council of the Netherlands (2001); and an International Agency for Research on Cancer monograph report (2001). The Netherlands report commented only on the cancer for which it thought there was possible risk (i.e., childhood leukemia).

A. Childhood leukemia

Degrees of confidence in causality by California reviewers: **99, 55, 75%**
IARC classifications by California reviewers: **Group 1, 2B, 2A**

None of the review panels has ruled out the possibility that there may be a slight risk of childhood leukemia associated with very high exposure levels of 50 Hz or 60 Hz magnetic fields. The concern arises because summary statistical analyses of epidemiological studies of residential exposures in children have reported a relative risk for leukemia of about 2 for average residential exposures exceeding 0.4 μT. As indicated by the summary statements below, no review panel has considered the evidence sufficient to conclude that there is a causal relationship (the degree of confidence in causality is supposed to reflect the probability that a causal relationship exists).

NRPB, 2001. "There is some epidemiologic evidence that prolonged exposure to higher levels of power frequency magnetic fields is associated with a small risk of leukaemia in children. In practice, such levels of exposure are seldom encountered by the general public in the UK. In the absence of clear evidence of a carcinogenic effect in adults, or of a plausible explanation from experiments on animals or isolated cells, the epidemiologic evidence is currently not strong enough to justify a firm conclusion that such fields cause leukaemia in children." (p. 164)

"In those studies in which measurements were made, the extent to which the more heavily exposed children were representative is in doubt, while in those in Nordic countries in which representativeness is assured, the fields were estimated and the results based on such small numbers that the findings could have been due to chance." (p. 163)

IARC, 2001. "IARC has now concluded that ELF magnetic fields are possibly carcinogenic to humans, based on consistent statistical associations of high level residential magnetic fields with a doubling of risk of childhood leukemia. Children who are exposed to residential ELF magnetic fields less than 0.4 microTesla have no increased risk for leukaemia." (Press release). The "statistical association between childhood leukaemia and power-frequency residential magnetic field strengths above 0.4 microTesla ... is unlikely to be due to chance, but may be affected by selection bias. Therefore this association between childhood leukemia and high residential magnetic field strengths was judged limited evidence for excess cancer risk in exposed humans." (summary conclusion).

Netherlands, 2001. "The committee concludes that these recent meta-analyses show a consistent association between relatively high measured or calculated magnetic field strengths and an increased risk of childhood leukaemia." (p. 40)

"The committee would emphasise that there is no known mechanism that could account for the association referred to above. The committee therefore sees no reason to modify its earlier conclusion that the association is not likely to be indicative of a causal relationship." (p. 40)

"It therefore remains the committee's belief that it is not likely that children (or adults) living near to high-voltage power lines are at risk through exposure to electromagnetic fields generated by those lines." (p. 40)

Other EMF scientists, 2001. The authors of a childhood leukemia study reporting a pooled analysis of multiple studies that has influenced recent review committees express some reservations themselves. They state, "The explanation for the elevated risk is unknown, but selection bias may have accounted for some of the increase" (Ahlbom et al. 2000). A recent editorial further states, "The evidence for an effect of ELF EMF on risk of childhood leukaemia remains unconvincing" (Ahlbom and Feychting, 2001).

Summary: The degrees of confidence expressed by the California reviewers seem to indicate that they believe a causal link between magnetic fields and childhood leukemia is likely. This conclusion is not consistent with the other recent summary reviews and by the scientists who have conducted EMF studies of childhood leukemia. Reviewers 1 and 3 gave higher IARC classifications than the classification, Group 2B, assigned by the IARC Committee.

B. Adult Cancers

Adult brain cancer

Degrees of confidence in causality by California reviewers: **98, 52, 70%**
IARC classifications by California reviewers: **Group 1, 2B, 2B**

Adult leukemia

Degrees of confidence in causality by California reviewers: **85, 52, 40%**
IARC classifications by California reviewers: **Group 1, 2B, 2B**

NRPB, 2001. Residential studies: "There is no reason to believe that exposure to electromagnetic fields is involved in the development of leukaemia or brain tumours in adults, although this possibility cannot be excluded." (p. 135)

Occupational studies: "No risk of brain cancer and leukaemia has been established with any confidence." (p. 158)

"The more recent cohort studies using better exposure characterisation for magnetic and electric fields either have not shown an association with leukaemia or brain cancer, or the association has been weak." (p. 167)

IARC, 2001. "No consistent evidence was found that residential or occupational exposures of adults to ELF magnetic fields increase the risk of any kind of cancer." (Press release)

Summary: The high degrees of confidence in causality expressed by the California reviewers are out of line with the conclusions of the recent, independent scientific reviews. In particular, there seems to be no scientific basis for considering the strength of evidence for adult brain cancer to be comparable to the strength of evidence for childhood leukemia, but that is what the California degrees of confidence would indicate. All three California reviewers gave higher IARC classifications for both adult leukemia and adult brain cancer than the classification, Group 3, assigned to both cancers by the IARC Committee.

IV. Neurological Disorders and Suicide

There have been fewer comprehensive reviews of the evidence for a possible association between magnetic field exposures and diseases other than cancer. For neurological diseases and suicide, however, few papers have been published since the release of the NIEHS Working Group Report (1998), and thus the extent to which the California conclusions are consistent with the deliberations of independent scientific experts is possible by comparing the California EMF Program report to the NIEHS Working Group Report.

A. Amyotrophic Lateral Sclerosis

Degrees of confidence in causality by California reviewers: **60, 60, 55**%
IARC classifications by California reviewers: **Group 2B, 2B, 2B**

The California report considered no paper published after the latest paper considered by the NIEHS Working Group. Nonetheless, each of the California reviewers gave ALS a higher IARC classification (consistent with the high degrees of confidence) than the classification, Group 3, assigned in the NIEHS Working Group Report. The ALS conclusion (i.e., inadequate evidence) in the NIEHS report was supported by 24 reviewers, with one abstention and 4 absences from the vote. Thus, the agreement of all three California reviewers for a higher classification is difficult to reconcile with the scientific evidence, and may reflect the lack of independence in the California deliberation process.

B. Alzheimer's Disease

Degrees of confidence in causality by California reviewers: **40, 20, 15%**
IARC classifications by California reviewers: **Group 3, 3, 3**

The California report considered one paper published after the latest paper considered by the NIEHS Working Group. The most recent study found no evidence for an association (the estimated relative risk was 0.74), so the general agreement between the California and NIEHS reports that there is insufficient evidence for an association is not surprising. The Alzheimer's Disease conclusion (i.e., "inadequate evidence") in the NIEHS report was supported by 23 reviewers, with one vote for a lower classification (i.e., "lack of evidence"), one abstention, and 4 absences from the vote.

C. Suicide

Degrees of confidence in causality by California reviewers: **49, 45, 45**%
IARC classifications by California reviewers: **Group 3, 3, 3**

The California report considered two papers not considered by the NIEHS Working Group. In one of these, a study of electric utility workers (Kelsh, 1997), risk was reported only by job title, and thus the study is of questionable value. This study reported increased risk of suicide in linemen,

but similarly increased risk was observed for all jobs involving field work (the comparison group was administrative, technical and clerical workers employed in offices). The study summarized in the second of these papers (van Wijngaarden et al., 2000) estimated cumulative magnetic field exposure levels from job histories and a job exposure matrix; of six different exposure time periods considered, including total lifetime exposure, a significant increase in suicide rate with increasing cumulative exposure was found only for exposure in the calendar year before the year of death. The van Wijngaarden et al. study was reviewed in the advisory committee report to the Health Council of the Netherlands (2001, p. 43). This report discounts the importance of the study because of methodological weaknesses that are common to occupational epidemiology studies (e.g., the same exposure level is assigned to all individuals with a given job title even though there can be considerable within-job variation in exposure levels, and there is no information on important potential confounding factors, such as history of addiction or psychiatric problems). The van Wijngaarden et al. paper suggests that magnetic fields might increase the risk of suicide by lowering melatonin levels, but as was noted by the Netherlands report, there is no good evidence that magnetic fields reduce melatonin levels in humans (in spite of considerable experimental efforts to demonstrate such an effect). The California reviewers agree with the NIEHS report in assigning an IARC classification of Group 3 to suicide. The NIEHS suicide conclusion (i.e., "inadequate evidence") was supported by 17 reviewers, with 6 reviewers voting for a weaker classification (i.e., "lack of evidence"), 2 abstentions, and 4 absences from the vote. The degrees of confidence seem high in comparison to the NIEHS vote, but this may reflect the reviewers' interpretation of the later two studies.

Summary: The only major discrepancy between the California report and the NIEHS report is for ALS. This discrepancy cannot be explained by a difference in the epidemiologic evidence considered. The agreement of the three California reviewers that EMF exposure is a possible cause of ALS is surprising, in view of the agreement within the NIEHS Working Group that there is insufficient evidence that magnetic field exposure cause ALS.

V. Reproductive Outcomes - Spontaneous Abortion (Miscarriage)

Degrees of confidence in causality by California reviewers: **80, 52, 65**%
IARC classifications by California reviewers: **Group 2B, 2B, 2B**

The degrees of confidence and the IARC classifications by the California reviewers are all higher than the IARC classification, Group 3, assigned in the NIEHS Report. The NIEHS conclusion (i.e., "inadequate evidence") was reached for both occupational exposure and for environmental exposure (i.e., residential exposures, including electric blanket use). For occupational exposure, the conclusion was supported by 22 reviewers, with 4 reviewers voting for a lower classification (i.e., "lack of evidence"), 1 abstention and 1 absence; for environmental exposure, the conclusion was supported by 23 reviewers, with 1 reviewer voting for a lower classification (i.e., "lack of evidence"), 1 abstention and 4 absences.

The higher classification by the California reviewers is due to the results of two unpublished California studies that suggested that high maximum magnetic field exposure levels were associated with spontaneous abortion (Lee et al., 2000; Li et al., 2000 - Appendices 5 and 6 of the California EMF Program report). This magnetic field exposure metric, i.e., maximum daily exposure level, has not previously been investigated in studies of miscarriage. Two of the California reviewers were authors of the first study (Lee et al., 2000), and the contributions of all three California reviewers were acknowledged in the second study (Li et al., 2000). These studies had a large impact on the classification by the California reviewers, in spite of the fact that on page 33 of the California EMF Program report the results of these studies are correctly identified as "hypothesis-generating data". As noted in the discussion of the first paper, the results of the two unpublished studies are inconsistent with the results of previously published papers (Lee et al., 2000, pp. A-137 through A-139 of the California EMF Program report). Both studies were handicapped by very poor participation (i.e., less than 50% of pregnant women participated in the measurements in both studies), and thus both studies have considerable potential for bias. The results on maximum magnetic field level need to be replicated in better studies conducted by independent investigators before this new proposed mechanism can be considered in determinations of the strength of evidence for causality for spontaneous abortion.

Summary: The high degrees of confidence in causality expressed by the California reviewers are out of line with the conclusions of previous scientific review groups. This apparently is due to their reliance on two unpublished studies of a novel magnetic field exposure metric in which they were directly or indirectly involved. These studies, however, were identified as "hypothesis generating", and need to be confirmed in independent investigations before results using the new proposed metric are included in assessments of possible adverse health effects associated with magnetic field exposure.

VI. References

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Appendix A

Cancer Appendix 1. Calculated fields versus measured fields: the Swedish study

Recent large epidemiological studies have shown no convincing evidence of increased risk of childhood leukemia associated with surrogate measures of magnetic field strength such as wire code category or proximity to power lines. Thus, the emphasis has shifted to examining risk associated with levels of magnetic fields determined by actual measurements (California report, p. 31). Calculated fields used in several Scandinavian studies have been considered to be equivalent to measured fields in several recent summary analyses of childhood leukemia risk. The calculated field for a residence is based on the physical relationship between a power line and the residence (i.e., data similar to those required to determine a wire code), as well as the electrical load carried by the power line in the year for which the magnetic field is being estimated.

The Swedish study of childhood leukemia (Feychting and Ahlbom, 1993) is by far the largest and most influential of the studies based on calculated fields. It is worth noting that the Swedish study matched controls to cases, and that the matching criteria guaranteed that the control lived in the same parish as the case and lived next to the same power line as the case. Thus, since the magnetic field was calculated for the same year for each case and matched control, the electrical load used in the calculation would be the same for each case and matched control (because the power line is the same). That is, differences between the calculated field for a case and matched control were the result only of differences in the physical relationship between the power line and residences of the case and control. Hence, in some sense, the comparison of calculated fields between a case and matched control is analogous to a comparison of wire codes.

Close examination of the Swedish study reveals contradictory evidence regarding the risk of childhood leukemia associated with actual measurements of magnetic field strength. Analyses based on actual measured fields in the Swedish study indicated that leukemia risk was lower in homes with high measured fields (Table 9 of Feychting and Ahlbom). This analysis has been discounted because measurements were available only for 62% of homes in the study, and because the measurements were considered to be less relevant to risk than were the calculated fields since the measurements were taken after leukemia was diagnosed. There are other analyses of the Swedish data that raise questions about the possible association of measured magnetic fields and childhood leukemia, however, and these analyses include all residences and cannot be discounted because of temporality.

The following table shows the percentage of residences in the Swedish study with measured field levels exceeding 0.2 μT and 0.3 μT by type (apartment versus one-family home) and location (Stockholm versus elsewhere in Sweden) of residence.

	Type of residence		Location of residence	
	Home	Apartment	Stockholm	Elsewhere
>0.2 μT	13%	28%	26%	12%
>0.3 μT	9%	19%	18%	8%

Clearly, actual measured magnetic field levels were much higher in apartments than in one-family homes and were much higher in Stockholm than in the rest of Sweden. In an analysis including all residences in the Swedish study, the odds ratio for leukemia associated with living in an apartment is 0.9 (95% CI, 0.4-1.9). Because controls were matched to cases by parish residence, an odds ratio cannot be calculated for Stockholm compared to the remainder of Sweden. However, 51% of the study base resided in Stockholm, while only 47% of the leukemia cases resided in Stockholm. Thus, analyses of both the type and the location of residence suggest that leukemia risk is inversely associated with measured magnetic field levels. Unless the higher magnetic field levels in apartments and in Stockholm were only a recent development, temporality of measurements is not an issue in these analyses.

It is also interesting that, in spite of the very large difference in measured magnetic field levels between apartments and one-family homes, there was no difference in calculated field levels between apartments and one-family homes. Thus, calculated fields obviously are missing important sources of magnetic field exposure in some residences.

Summary: The decision by some review groups to give studies based on calculated magnetic fields the same weight as studies based on actual measurements of magnetic fields requires additional consideration.

Appendix B

Cancer Appendix 2: The importance of a plausible mechanism

An interesting parallel can be drawn between the epidemiologic evidence that magnetic field exposure causes childhood leukemia and the epidemiologic evidence that *in utero* exposure to ionizing radiation causes childhood leukemia. The latter exposure is usually listed as one of the few accepted risk factors for childhood leukemia, but as noted below, the epidemiological evidence is not particularly strong.

The first two studies (both case-control studies) to report an association between childhood leukemia and magnetic field exposure (Wertheimer and Leeper, 1979; Savitz et al., 1988) also evaluated the risk for cancers other than leukemia. Interestingly, evidence of risk was found for all types of cancer in both studies. Wertheimer and Leeper evaluated the risk for the wire code at both the house resided in at the time of birth and the house resided in at the time of death. The odds ratios for high wire codes compared to low wire codes for leukemia, lymphoma, central nervous system (CNS) tumors, and all other cancers were as follows:

	Leukemia	**Lymphoma**	**CNS**	**Other**
WL Birth	2.3	2.5	2.4	2.4
WL Death	3.0	2.1	2.4	1.1

Savitz et al evaluated risk for a single house, and the odds ratios for high wire codes compared to low wire codes for leukemia, lymphoma, CNS tumors, soft tissue sarcomas (STS), and other cancers are as follows:

	Leukemia	**Lymphoma**	**CNS**	**STS**	**Other**
Savitz	2.8	3.3	1.9	1.7	1.6

The similarity in the magnitude of the odds ratios in both studies for all types of cancer is puzzling, and either the data suggest that magnetic fields cause all types of cancer, or the uniformity of odds ratios provides empirical evidence of bias in these studies. Both studies had major flaws in design that could have resulted in bias. Subsequent studies of childhood cancer have found the potential for risk associated with magnetic fields only for leukemia, and the magnitude of the relative risks indicated has generally been smaller than those reported in the first two studies.

A similar situation exists with regard to epidemiological studies of *in utero* ionizing radiation. The first case-control study (Stewart et al., 1958) of *in utero* ionizing radiation reported the following odds ratios:

Leukemia	Lymphoma	CNS	Neuroblastoma	Wilms Tumor	Bone	Other
1.5	**1.4**	**1.4**	**1.5**	**1.6**	**1.1**	**1.5**

A later case-control study (MacMahon, 1962) found similar results, reporting an odds ratio of 1.5 for leukemia and an odds ratio of 1.5 for all other childhood cancers.

The similarity in the magnitude of the odds ratios in both studies is, once again, puzzling, and suggests the possibility of bias. Even though ionizing radiation is known to induce some cancers in adults, not all types of cancer are equally radiogenic. An extension of the second study (Monson and MacMahon, 1984) reported odds ratios of 1.5 for leukemia and 1.1 for all other cancers. A number of cohort studies have been performed to investigate the associations, and virtually all of these have found no evidence of childhood cancer risk following *in utero* radiation, even for leukemia (Boice and Miller, 1999; UNSCEAR 2000). The absence of an increase in childhood cancer following *in utero* exposures among the Japanese survivors of the atomic bombings is noteworthy.

In spite of the fact that the epidemiological evidence for causality of the association between *in utero* irradiation and childhood leukemia is equivocal, *in utero* ionizing radiation is usually listed as an established risk factor for childhood leukemia. The existence of a plausible biological mechanism (based on known physical properties of ionizing radiation) is the reason that *in utero* irradiation is accepted as a risk factor, coupled with the fact that exposures during childhood and adulthood are clearly leukemogenic.

Summary: The lack of experimental data (biological or physical) supporting a plausible mechanism for induction or promotion of cancer by magnetic fields is an important reason why the weak epidemiological evidence has not been sufficient to convince the scientific community of a causal link between magnetic fields and childhood leukemia.

Appendix C

Reproductive Outcomes Appendix 1

Comments on "A nested case-control study of residential and personal magnetic field measures and miscarriages" by GM Lee, RR Neutra, L Hristova, M Yost, RA Hiatt

This study was conducted among participants in a prospective study to investigate possible associations between source of drinking water and spontaneous abortion. Pregnant women were recruited when they called to schedule their first prenatal visit. All English- and Spanish-speaking women at least 18 years of age and in the first 13 weeks of their pregnancy were eligible. The participation rate cannot be calculated from the published paper (Epidemiology 1998;9:126-33), because ineligibility and refusal were reported together. That is, of 7,881 women calling to schedule their first prenatal visit, it is reported that 6,179 (78.4%) were eligible and agreed to participate. Of these 6,179 initial responders, 268 women were no longer pregnant when reached for the enrollment interview and 569 women did not participate. Thus, of 7,613 women approached (i.e., excluding the 268 women from the initial total), 5,342 (70%) participated by completing the enrollment questionnaire. It seems likely that the participation rate (assuming ineligibles were enumerated and removed from the above calculations) was probably less than 80% in the prospective study of drinking water source.

At the time of the enrollment questionnaire for the prospective water source study, women (4-13 weeks gestation, with a mean of 8 weeks gestation at enrollment) were recruited for the prospective component of the magnetic field study. For this prospective sub-study, a random sample of 531 women was chosen and these women were asked to participate in magnetic field measurements at around 12 weeks gestation. Only 219 women (41%) participated. These women, as well as women in the larger nested case-control study, also were asked to participate in magnetic field measurements at around 30 weeks gestation. Of the 219 women, 18 were eventually cases (i.e., their pregnancies terminated at 20 weeks gestation or less) and 201 were eventually controls. Only 10 of the 18 cases (55%) participated in the second measurement component, while 166 of the 201 controls (83%) participated in the second measurement component.

For the nested case-control study, medical charts were reviewed at around 25-30 weeks gestation to identify cases (pregnancies terminating at 20 weeks gestation or less) within the entire cohort involved in the water source study. All 328 women with a reported miscarriage and a random sample of 806 women who were still pregnant were identified as potential cases and controls. All of these women were asked to participate in a magnetic field measurement study at around 30 weeks gestation. Only 167 (51%) of cases and 384 (48%) of controls eventually participated.

The participation rates in both components of the magnetic field study were extremely low, especially considering that the participation rate in the larger water source cohort study within which the magnetic field study was conducted was probably already less than 80%. For the nested

case-control measurements at around 30 weeks gestation, participants were excluded if they had moved between the first trimester and the time of the measurements; of the 177 cases (prospective and case-control women combined), 22 (12%) were thus excluded, and of the 550 controls, 41 (7%) were thus excluded.

With such low participation rates at all stages of the study, selection bias is a potential problem. Evidence of such bias was observed in the case-control component for the one measure of exposure that could be evaluated (i.e., wire codes). Prospective studies are not immune to problems with selection bias. If participation is related to a factor that is associated with both risk of miscarriage and magnetic field exposure, then bias can be introduced.

The authors report a poor correlation between the two measurements for the 176 women in the prospective sub-study who participated in both measurement components. This suggests that personal measurements on a single day do a poor job of characterizing a woman's maximum magnetic field exposure level.

The timing of the case-control measurements could also have introduced bias, because the cases were no longer pregnant when measurements were made at 30 weeks, while the controls were still pregnant at the time of the measurements. If pregnant women were more careful, and less likely to spend time around electrical appliances or other sources of magnetic fields, then this could have created an artifactual difference between cases and controls in personal measurements. In fact, there were no differences between cases and controls in residential measurements of magnetic field levels; the only differences were related to the personal measurements.

Although the prospective component of the study is represented as confirming the case-control study, the prospective study showed 2-fold increases in miscarriage rates for increased levels of all measurements of magnetic field strength (i.e., residential as well as personal measurements). This suggests that a different source of bias may have been at work in the prospective component of the study.

Summary: Low participation rates raise the possibility of bias. Residential measurements of magnetic fields were not associated with early miscarriage but personal measurements were, suggesting a bias, possibly because cases (not pregnant at the time of measurements) and controls (pregnant at the time of measurements) behaved differently at the time of the personal measurements. The absence of a dose response relationship with maximum magnetic field level raises questions about the causal nature of the association. Results need to be replicated in independent studies.

Appendix D

Reproductive Outcomes Appendix 2

Comments on "A population-based prospective cohort study of personal exposure to magnetic fields during pregnancy and the risk of spontaneous abortion" by D-K Li, R Odouli, S Wi, T Janevic et al.

This study was performed to confirm the finding in the prospective sub-study component of the previous California study (Lee, et al., 2000) that time-weighted-average magnetic field exposure above 2 mG conveyed increased risk of spontaneous abortion. All women submitting urine to the San Francisco or South San Francisco facilities of the Kaiser Permanente Medical Care Program for a pregnancy test were given a flyer describing the purposes and procedures of the proposed study of magnetic fields; the flyer included a self-addressed refusal postcard. All English speaking women with a positive pregnancy test who didn=t return the postcard were eligible for the study if their gestational age at the time of the pregnancy test was 10 weeks or less, and they intended to carry the pregnancy to term.

Of 2729 eligible women identified, only 1063 (39%) completed the enrollment questionnaire and the magnetic field measurements. Assuming that some pregnant women returned the refusal postcard, the actual participation rate is even lower. An additional 73 women were excluded because of problems with their measurements or activity diaries for the measurement day, and 21 women were excluded from analyses because of missing data. Thus analysis was based on 969 women (i.e., 36% of the total eligible). The extremely low participation rate raises serious questions regarding the possibility of bias in this study.

Analysis of time-weighted-average magnetic field strength did not confirm the results of the previous study. Significant relative risks were reported for maximum magnetic field levels exceeding 16 mG. The authors state that they expected *a priori* that maximum magnetic field level would be a better measure for detecting a biologic effect. The relative risk observed for spontaneous abortion with maximum magnetic field level does not increase, however, with increasing maximum level above 16 mG. The authors do not explain why the lack of a dose-response should have been expected for biologic effects.

The 16 mG level used in the statistical analyses was chosen on the basis of examining the observed relative risks by decile of maximum level. There is no adjustment of the reported significance levels for this data-driven, *post hoc* selection of cut-point for the analyses.

Almost 75% of women in the study experienced maximum magnetic field exposures in excess of the supposed threshold level of 16 mG. This suggests that there may be something unusual about the minority of women who do not experience such high levels. Examination of the Appendix to the paper suggests that they may have been more concerned about how their behavior might impact on the outcome of their pregnancies. The women with low maximum magnetic field measurements were less likely to smoke, consume alcohol, drink coffee, use a Jacuzzi, or drink tap

water during their pregnancy. They were also less likely to be single, less likely to have worked, less likely to carry loads, and less likely to exercise strenuously. While the differences are not large individually, they, along with the potential for bias because of the very low participation rate, raise questions about the extent to which the reported differences in spontaneous abortion rates can be attributed to differences in maximum magnetic field levels.

Summary: Hypothesis generating study that requires confirmation in other settings. The low participation rates and the consistent differences between women with low versus high maximum magnetic field measurements in numerous potential risk factors for miscarriage suggest the need for caution in interpretation of findings. The absence of a dose response relationship with maximum magnetic field level raises questions about the causal nature of the association. Results need to be replicated in independent studies.

Appendix E

IARC Classifications

Group 1: The agent (mixture) is carcinogenic to humans. This category is used when there is sufficient evidence of carcinogenicity in humans. There are 87 agents classified in Group 1, e.g., asbestos, benzene, radon.

Group 2 (two classifications):

Group 2A: The agent (mixture) is probably carcinogenic to humans. This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. There are 63 agents classified as Group 2A, e.g., formaldehyde, nitrogen mustard, trichloroethylene.

Group 2B: The agent (mixture) is possibly carcinogenic to humans. This category is used when there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals. There are 235 agents classified as Group 2B, e.g., phenobarbitol, saccharin, coffee.

Group 3: The agent (mixture, or exposure circumstance) is unclassifiable as to carcinogenicity in humans. This category is used most commonly for agents, mixtures and exposure circumstances for which the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals. There are 483 agents classified as Group 3, e.g., cholesterol, coal dust, fluoride.

Group 4: The agent (mixture, exposure circumstance) is probably not carcinogenic to humans. This category is used for agents or mixtures for which there is evidence suggesting lack of carcinogenicity in humans and in experimental animals. Only one agent (caprolactum) is listed as Group 4.

Appendix References

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Answers to questions on the 3rd draft of the California EMF risk evaluation

Nancy Wertheimer 9/4/01, University of Colorado, 1330 Fifth Street, Boulder, CO 80302

1. A wise decision I think. The epidemiologic evidence combined with a small but growing number of positive laboratory findings not easily explained by present biological knowledge is now sufficiently strong to ask biologists to look beyond the present knowledge.

2. (In line 6, change you're to your) My own prior degree of confidence in risk attributable to EMF exposure was extremely low at the time of our first report in 1979. It has increased a great deal since then, based largely on the weight of the cumulative epidemiologic evidence (which includes more than the occupational and residential studies: For instance a survey of electric blanket use and childhood leukemia might show considerable strength and consistency in its evidence of risk. Such use showed an O.R. of 1.5 in the Savitz study, 2.25 in the Linet data, and 7.0 in the London study (based on only 8 discordant pairs).

   The lack of a dramatic change since the introduction of electricity is not a concern to my mind, for several reasons including:

   (A) Milham's article with evidence that rural electrification **did**, in fact, introduce a new cause of leukemia in rural children. **This paper should be** cited in your report. I attach a first sheet of the paper giving the reference.

   (B) My own knowledge, from years of field work, about how much prolonged exposure to magnetic fields has been **decreased** over the years by technologic changes in power delivery, etc., such as: (1) Use of non-conductive plumbing pipes and connectors. (2) Increased use of 240 volt (vs 120 volt) appliances (The latter can produce ground currents in house plumbing,; the former generally will not do so), (3) Installation of a plus and a minus live wire in the service to a house instead of a single live wire (the latter means all appliance-caused ground currents will add their effects; the former means appliance-generated currents can cancel each other, (4) Use of three-phase wires to deliver higher current loads, thus tending to mitigate any increase in fields due to increased loads, (5) Increasing the voltages used, thus allowing a smaller current to deliver a larger amount of power).

3. I agree. No comment.

4. I agree. Given our lack of understanding of EMF "dose", and of the host- and environmental-cofactors needed to produce effects, even the most carefully done animal work may be invalid.

5. I agree. Most epidemiologists aren't usually asked to think about epidemiologic evidence obtained where a "dose" can't be defined with any confidence -- and where the problem is made worse because we don't know if dosage is cumulative, or at what time prior to diagnosis the exposure should be evaluated. This is not a subject where traditional expectations based on cearcut independent variables and initiation of cancer can be applied.

6. I agree. In fact I expect lack of specificity, based on the likelihood that EMF is an enhancer of the carcinogenic process rather than an initiator.

7. Based on the accumulated epidemiologic evidence, I think your assessments are well-balanced. Those strongly oriented against an effect by our lack of physical understanding may see you as too pro. I don't.

8. I think your present wording is adequate.

Overall, this is a very thoughtful and useful risk analysis.

Page #	Table #	Line # or Comment # in Table	Comments
31-32	3.1.1		Auvinen (reference page 1 attached) should be cited for its analysis of alternative exposure metrics, as pertinent to this table.
38		5.3, line 5	Two published replications of Liburdy are now published and should be cited: Blackman and Ishido--both references*attached here. page 1 only
90	8,2.2	3rd point	Given the major demographic changes in L.A. over the years in question, I am skeptical that the 1984-91 Preston-Martin cases make good controls the 1980-87 Leukemia cases.
94	8.2.6	A-1	I don't see how a flawed negative study is seen as an argument **against** causality. The positive findings in some second co-authored article have never been used as an argument for more than the flawed nature of the study-- and were **not** co-authored by Fulton, I'm reasonably sure.
95	8.2.7	A-1	Earlier studies **did** show evidence of a threshold at 3 mG. Tomenius saw no risk until that level; Savitz reports that O.R. was high at 3 mG. but imprecise (p37). The Ahlbom data show a high risk at 3 mG. In fact in Greenland's metaanalysis it- is clear that most stuides showed elevated ORs at 3mG but not at 2-3 mG. The A-1 statement is, I think, wrong.
97	8.2.8	A-2	How is the Swedish study inconsistent with other studies? Either the total sample or the one--family data show an increased risk at 3+ mG.
120	9.2.6	A-2	Change "failed to show" to "showed".

Hello Dr. Collins.

I suggest you visit the American Physical Society web site at:

http://www.aps.org/statements/95.2.html

There you will find that these issues have been examined by competent experts with no administrative motivation pro or con.

The National Academy of Sciences has concluded that power lines are not harmful, so it is unclear why the investigators on this project continued through the 1990's and into the 21st century making a lot of work about "EMF".

See: http://books.nap.edu/catalog/5155.html?onpi_newsdoc103196

I did not read the entire text of the report, which was posted in a disconnected collection of tiny files, too time-consuming to download individually.

However, what I did read in the study did not inspire confidence. There seemed to be primarily a political agenda, and no real effort at objectivity as opposed to argumentation.

For example, in the "Science Abstract", I find such absurdly uncientific images as,

> "EMFs are complex physical agents, which exhibit many distinct properties. Sometimes they may be compared to a mixture of ingredients, of which one or more may be hazardous and others harmless."

Is there anything in the real world that does NOT "exhibit many distinct properties"?

Also, the "scientists" did not seem to be identified. Was this to discourage fabrication or irresponsibility?

The chapter on "biophysical issues" was unbelievable: What does "For Causality" versus "Against Causality" mean? Where was there any biophysics invoked?

I have two suggestions:

1. I suggest that you issue the Executive Summary as the report, and supply the rest of the work on demand, as a set of links or footnoted references.

2. I suggest you eliminate all reference to "epidemiology": This is not science but statistics. For example, if I claimed sweating was dangerous and potentially fatal, all I would need do was construct a statistical sample of healthy athletes and emergency room patients, some suffering fever or infection. There then would be, in the group doing more sweating, a statistically significant probability that sweating would be followed shortly by death.

If I could not calculate statistical significance, I would include more and more of the healthy + sick sweaters until I achieved significance.

Is this the California Health Plan for EMF "research" on many distinct properties?

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Dear Drs Collins and Neutra

Thank you for sending me a copy of your report on evaluation of the possible risk of electromagnetic fields. As I stated to Raymond in an earlier communication, I have been extremeley busy in the last month on two items which took higher priortiy:
(1) ensuring that cancer patients being treated without a hitch with protons here in the Boston area continued to be treated without any interruption as the Harvard Cyclotron closes and the Northeast Proton Therapy Center comes into operation and
(2) ensuring that the 2nd edition of "Rick-Benefit Analysis" got printed in time for fall classes. (printed copies arrived this week)

Since you have today as a deadline I have only a very quick response.

This study has clearly involved an extraordinary amount of work on a difficult subject which has never before had such attention. But I believe that it is flawed (although perhaps not beyond redemption).

A.) You have correctly discussed a number, $5 million that people seem to be prepared to pay fore reducing risks when they are only statistically detemined. This is now used by US EPA. However, there are huge variations in this as noted in Chapter 5 of the book noted above. This of course is related to the discussion of "social justice". However there are major problems with application of that concept. All too often it is raised, and misused, by people who want to decry proper logical analysis, or get their own way by pretending to help others. often it is raised by uninformed "do-gooders" . The important issue, all too often forgotten or ebven tampled on, is what do the people who are the victims of injustice want and need?

People in poorer areas should be asked the direct questions.

We have $10 million to spend to improve your lot. Do you want:
(1) better busservice,
(2) power lines underground
(3) cheaper electricity

My experience with hearing such questions discussed by those concerned (especially in a couple of public hearings that I have attended in California) is that no one wants (2) at the expense of (1) or (3) All the discusssion that I see in the document is appallingly patronizing.

B. Sir Austen Bradford Hill, in his famous discussion of the attributes of an "association" that might lead someone to assign causality, mentioned biological plausibility. But he also warned that one should consider this with great care because a scientific heresy of today may become the scientific truth of tomorrow. Following his advice, you have rightly considered it appropriate to discuss the statistical associations as if they were causal and see where that discussion leads. But to consider societal action based on that assumption needs far greater care.

I make two major points that seem to have been neglected by your reviewers. (i) Since the possibility that electromagnetic fields at low intensities (milliGauss) may cause was raised about 25 years ago, there has been intensive discussion of possible mechanisms. NOT one mechanism has been proposed that is not in contradiction with the well established laws of electricity and Magnetism. (This flat statement seems in contradiction to the

statement made on page 305 under the heading "biophysics" but I stand by it). (ii) NOT one epidemiologist has stood up and said, my result is so reliable and my interpretation so correct, that I am prepared to say that there is something new going on that is contradiction to the work of Maxwell, Einstein and Dirac.

Although this does not prove that no such effect exists, it is enough to ensure that scientists who understand and regularly use the work of the three above mentioned physicists would ALL assign degree of confidence that Adult Leukemia or Female Breast Cancer can possibly be caused by electromagnetic fields at the milliGauss level is MUCH less than 10% and probably zero.

Of course the expression "10% possible" is a peculiar expression in itself and open to various interpretations. If it is to be used in a policy consideration it must be well defined - and is not well defined in your document. I would roughly define 10% possible in a pragmatic way, by considering the sattement as part of a caluclation of the risk using Bayes theorem. 10% possible would mean a prior probability of 0.1, to be modified as appropriae by epidemiological or other studies.

That your three reviewers all say that it is greater than 10% possible (page 303) can either be due to: (1) they have a different interpretation of the words "10% possible" (2) they are not knowledgeable about the work of Maxwell, Einstein and Dirac. (I note that the letters EMF was known to all of them as "Electro Motive Force" and its use otherwise shows a lack of appreciation of the historical context) (3) they are influenced by the factual statement about funding on the bottom of the left hand column of page 306

This then says something about the choice of reviewers which clearly is NOT either random, or chosen accoring to their skills as expressed in public recognition (degrees, membership in NAS, etc). If one estimates the number of scientists who would assign zero, and the number who would say, yes it is possible, from the utterances of the members of the bioelectromagnetics society, one would find a dichotomous distribution. Some meetings would be unanimous in saying zero, other meetings might be unanimous in saying greate than 10%. I suspect that a poll of the whole membership would find at least half would say close to zero (according to my interpretion of what possible means) If one took membership of NAS and asked for a quotable statement, (rather than an anonymous one in which the reptation is not at stake) the number saying zero would be more than half.

In interpreting Sir Austen Bradford Hill's remark one must be aware that the laws of physics and the laws of biology are different. The fundamental laws of phyics are relatively few and are well grounded. The laws in biology are less based upon fundamentals; they are more numerous and can be considered more as postulates to be repeatedly changed and modified. I suspect that the decision to ignore the fundamental laws of physics is based on a misunderstanding of how well established they are.

I repeat, as I have done before, the introductory words of the elementary undergraduate text on biology of the cell authored by one of my colleagues. "Cells obey all the laws of physics and chemistry." Anyone proposing a model that is in violation of this has a major responsibility.

B. All the epidemiological discussions are peculiar. Often epidemiological studies have to be based upon a simple exposure metric: 'Yes he worked here/ no he did not work here" . Once one goes beyond such a dichotomous statement and compares to a measured something, one should do it right.

Very fundamental issues of symmetry state clearly that at low intensities any effect of magnetic fields must vary as the square of the field. (or perhpas for alternating fields as the average of the mean square over a long periods (as stated in my earlier comments). I do not know of any study that does this. When I plot, for example, Savitz data, I find that it seems to vary as the square root or be a step function. This is a very strong suggestion that the true cause is nothing to do with the field at all but perhaps there is something systematically wrong about the control group. I cannot find in the epidemiological studies listed any indication that the authors have considered this.

Assocated with this problem is the necessity of discussing the dose reponse in more generality than in the particular study. This is best done in the framework of a simple algebraic model. I will for the moment (temporarily and relucatntly) concede that the work of Maxwell, Einsten and Dirac is incomplete, but one needs to follow simple arithmetical rules (unlike Alice do not ask for two impossible things at once). I would prefer to assume that the effect must vary as B squared, and then predict, from the claimed result of the epidemiological studees the effect at 1 Gauss. Then one can ask the simple question : is that consistent with experience of high exposures? I can find no such discussion in this document. My belief, based on personal experience (i.e. I am still alive and welll even though I have spent many hours in 15 kiloGauss) is that there is no consistency and from this reason alone the association with low intensity fields is unlikely to be causal.

C. In 1990 it was suggested by the US EPA staff that EMF be considered a "Class C Carcinogen" In 1991/2 this was extensively reviewed by a committee assembled for the purpose and , inter alia, it was agreed that the IARC classifications discussed on page 307 section 21.3 were NOT appropriate to the discussion of low intensity electromagnetic fields, and attempts to use such a classfication would lead to misunderstanding. Thus unless this is VERY carefully explained, (which so far it is not) the section 21.3 is misleading.

I hope that these quick comments are helpful

The connection between power lines, EMF, and the chronic conditions that you mention are actually well known and well documented, if you look at the right connections and documentation.

EMF is well documented to cause electrical currents in the metal in people's mouth(such as amalgam dental fillings, metal crowns, braces, etc.). And this is documented to cause movement of high levels of such metals, especially mercury into the oral cavity, blood, brain, etc. www.home.earthlink.net/~berniew1/galv.html

(the daily exposure in such situations is documented to be above Gov't health standards-see below)

And the mercury so depositied is well documented to cause at least 40 serious chronic health conditions(over 1000 medical study references documenting this all at: www.home.earthlink.net/berniew1/amalg6.html or www.home.earthlink.net/indexa.html

Actually it is known and well documented that:
1. power lines produce electric and electromagnetic fields.
2. both types of fields have adverse physical effects documented in
...animal and human studies-
affect calcium homeostasis in cells, melatonin levels, indirect known
...effects through influence on toxiic metals in the body. (these all
...have well documented adverse effects)
3. EMF causes galvanic currents in metals in the body, such as amalgam
...fillings, documented to
cause large increases in mercury in the body and brain
....(www.home.earthlink.net/~berniew1/galv.html)
4. mercury at such levels documented to cause at least 40 serious chronic
...conditions
(www.home.earthlink.net/~berniew1/amalg6.html or
...../damspr3.html

Using the well known and accepted rules of logic, this clearly implies that EMF from power lines is a part of synergistic effects that cause more than 40 chronic conditions.

The fact that population based epideological studies are difficult to draw firm conclusions from, doesn't mean there isn't a huge amount of research already documenting known adverse effects if you look at studies of the more managable parts that are easier to control, and put them together.

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